G16·06 JY48

DOMINA RERUM SAPIENTIA

Library
of the
Academy of Medicine,
Toronto.
4605

Presented by

The Society.

Digitized by the Internet Archive
in 2010 with funding from
University of Toronto

http://www.archive.org/details/proceedings1887newy

Path. Trans.

PROCEEDINGS

OF THE

NEW YORK

PATHOLOGICAL SOCIETY

FOR THE YEAR

1887

PRINTED FOR THE SOCIETY
1888

4605

LIBRARY
AUG 2 2 2001
UNIVERSITY OF TORONTO

LIST OF OFFICERS AND COMMITTEES FOR THE YEAR 1887.

President,

T. Mitchell Prudden, M.D.

Vice-President,

William P. Northrup, M.D.

Secretary,

Wesley M. Carpenter, M.D.

Treasurer,

John H. Hinton, M.D.

Editor,

John C. Peters, M.D.

COMMITTEE ON ADMISSION AND ETHICS.

J. C. Peters, M.D.,
Walter Mendelson, M.D.,
H. Marion Sims, M.D.,
L. Emmet Holt, M.D.,
W. H. Porter, M.D.

COMMITTEE ON PUBLICATION.

George C. Freeborn, M.D.,
William P. Northrup, M.D.,
John C. Peters, M.D., *Editor*,
W. M. Carpenter, M.D., *Secretary*,
J. H. Hinton, M.D., *Treasurer*.

COMMITTEE ON MICROSCOPY.

George C. Freeborn, M.D.,
J. H. Thacher, M.D.,
W. H. Porter, M.D.

THE MIDDLETON-GOLDSMITH LECTURES.

By M. ALLEN STARR, M.D., Ph.D.,

PROFESSOR OF NERVOUS DISEASES, NEW YORK POLYCLINIC.

MULTIPLE NEURITIS AND ITS RELATION TO CERTAIN PERIPHERAL NEUROSES.

Lecture I.

Mr. President, Ladies and Gentlemen of the Pathological Society :

In inaugurating the course of lectures which bears the name of the founder, it is becoming that some expression of gratitude should be made to Dr. Middleton Goldsmith. This can hardly be associated with any form of personal encomium, since that would be as distasteful to him as it is needless before this Society, which owes its existence in part to his exertion. It is proper, however, to emphasize our appreciation of the motives which have led to the establishment of this lectureship. Every one knows that in the rapid progress of medical science, in the great and needful division of labor which that progress has made imperative, there is offered to each individual worker a vast mass of facts which cannot be easily grasped or even readily comprehended. To classify these facts, to weigh their importance, to draw logical conclusions from them, and to present these conclusions in a clear and accessible form is a work of no little magnitude, but of absolute necessity. To accomplish this specialists are at work in every department, and it may be well to emphasize the fact that it is the necessity for their labor, rather than any temporary fashion, that has led to that specialization in medical science which is so often harshly criticised. But special studies lose half their value if they are not made generally available. And it is just here that a lectureship such as this has its use. It enables the results of individual labor to reach beyond the specialist, to be placed before the general practitioner in a simple but comprehensive way. There is, therefore, no means by which an individual can make his influence more widely and permanently felt than by the endowment of such a foundation. For when his personal activity has of necessity ceased with the natural termination of an honorable career he may be sure that, yearly, facts and results are proclaimed in his name of immeasurable service to his successors and of benefit to the profession.

And now, before proceeding to the subject which is to occupy our attention for a time, let me express my obligation for the honor which this Society has conferred in inviting me to inaugurate this course of lectures. It is an honor to which I have no claim, and the duty incurred is one which I feel incapable of performing satisfactorily. I must beg your indulgence in the performance of the task you have laid upon me, and if I succeed in eliciting your interest I know that it will be because the subject we are to study together has a novelty which may compensate for the defects of its presentation.

The discovery of a new disease is never made suddenly. It is a gradual process, and certain stages in the progress toward its complete recognition may be observed. There is first the period of clinical observation, when isolated cases of an unfamiliar and mysterious affection are recorded as curiosities. To this succeeds the period of diagnosis, when, by a comparison of the now numerous cases, a clinical picture of the disease is gradually filled out. In this stage there is much to impede the progress of discovery. For not content with an analysis of symptoms, and a grouping of cases, the majority of observers offer theoretical explanations of the nature of the new affection, and an element of speculation enters which often obscures the facts. There is, however, a real progress in this period, for it is characterized by inductive reasoning from fixed data, and as a result reliable conclusions are reached which make a diagnosis possible. The third period is that of pathological discovery, when the morbid changes lying at the basis of the disease are accurately ascertained. In this stage erroneous theories are eliminated, true explanations for various symptoms become self-evident, and the exact nature of the affection is determined. The disease has now a status of its own. And at first this might seem to be the final stage in the progress of discovery. But it is not. There remains a period of etiological classification, when conditions, formerly supposed to be dissimilar, are found to have a common basis, when the pathological changes are ascertained to be the same although the clinical pictures have varied, and when classification of the various forms is rendered possible and a definition of the disease is reached.

Such a gradual advance toward general recognition is well illustrated by the history of multiple neuritis, which is to engage our attention at the present time.

I think we may claim that one of the first cases presented was by an American physician, Dr. James Jackson, of Boston, in 1822.[1] In a paper "On a Peculiar Disease Resulting from the use of Ardent Spirits," which he named arthrodynia, he gives a most graphic picture of what we now know to be one form of multiple neuritis. He says, "This disease comes on gradually. It commences with pain in the lower limbs, but especially in the feet, and afterward extends to the hands and arms. The hands may be affected first in some instances, and in all cases in an advanced state the pain is more severe in the feet and hands than in the upper part of the limbs. The pain is excruciating, but varies in degree at different times. It is accompanied by a distressing feeling of numbness. After the disease has continued a short time, there take place some contractions of the fingers, and toes, and inability to use these parts freely. At length the hands and feet become nearly useless, the flexor muscles manifesting, as in other diseases, greater power than the extensors. The whole body diminishes in size, unless it be the abdomen, but the face does not exhibit the appearance of emaciation common to many visceral diseases. The diminution is especially observable in the feet and hands; and at some time the skin of these parts acquires a peculiar appearance. The same appearance is noticed in a slighter degree in the skin of other parts. This appearance consists in a great smoothness and shining with a sort of fineness of the skin. The integuments look as if tight and stretched, without rugæ or wrinkles, somewhat as when the subjacent parts are swollen; but the skin is not discolored. Yet in this disease there is not any effusion under the skin, and the character which this assumes arises from some change in the organ itself. The most characteristic symptoms are manifested in the limbs, but the pain is not limited to these—and other symptoms are exhibited in other parts. The pain sometimes shoots suddenly up one or both legs, and in one case it frequently passed up the back and then forward to the pit of the stomach. The functions of the stomach are always impaired. The mind is weakened. Sleep is prevented by pain. I believe that this disease is always fatal when the use of spirituous liquors is not abandoned before the powers of the digestive organs are greatly impaired."

It is hardly possible, even at the present day, to add to this description, which portrays in strikingly vivid language the main features of one form of multiple neuritis.

---

[1] New England Journal of Medicine and Surgery, vol. xi., p. 351.

The next observations of importance in establishing a clinical picture were made by Magnus Huss, who, in his work upon chronic alcoholism, in 1852, gave a very complete description of alcoholic nervous symptoms, dividing the cases into paralytic, anæsthetic, convulsive, epileptic, and hyperæsthetic forms. But not content with the clinical side, he advanced to pathological hypothesis, and ascribed all the various symptoms to lesions in the central nervous system. His description was amplified and completed by Lancereaux, in 1864, in an article upon alcoholism in the "Dictionnaire Encyclopédique des Sciences Médicales." But the pathology of the disease was still a matter of speculation.

In 1855 the great work of Duchenne was published,[1] and in it a number of cases are recorded which we now recognize as multiple neuritis. In these cases there were sensory disturbances, consisting of pain, numbness, and loss of sensation; motor disturbances, consisting of paralysis, with atrophy, especially marked in the distal parts of the extremities, and attended by a loss of faradic contractility in the paralyzed muscles; and cyanosis, coolness, and increased sweating in the affected limbs. Duchenne grouped these cases together under the title "Paralysie Générale Spinale Subaigüe Ascendante," because he thought a gradually advancing lesion in the spinal cord, from below upward, would explain the symptoms. It is true that he found no macroscopic change in the cord in the only case in which he made an autopsy. But when, under the leadership of the French school, from 1860 to 1865, the microscopic appearances in nervous lesions began to be studied, the hypothesis of Duchenne at once appeared to be verified, for it was found that such symptoms as numbness, pain, and anæsthesia were associated with lesions of the posterior columns of the spinal cord. And it was also proven that atrophic paralysis was caused by a degeneration of the ganglion-cells of the anterior gray horns of the cord, not necessarily visible to the naked eye. It seemed an easy step to the conclusion that when these symptoms occurred together the entire spinal cord was the seat of disease, and that wherever they occurred a spinal lesion was progressing. The pathology of this form of paralysis appeared to be definitely ascertained, and for many years the fallacy of such a conclusion was not detected. All atrophic paralysis was invariably referred to spinal lesions, because spinal lesions may cause atrophic paralysis.

---

[1] Electrization localisée.

But facts rarely accommodate themselves permanently to theories, and after a time a mass of very unwieldy facts began to accumulate. Cases of atrophic paralysis without spinal lesion were observed, and these threw doubt upon the theoretical pathology. The period of true pathological observation had begun, and gradually went on to completion. In 1864 Dumenil reported the following case, which deserves to be cited, as it is the first in which an autopsy established the existence of a wide-spread disease in the peripheral nerves as a cause of sensory, motor, and trophic symptoms.

Observation I.—A tailor, aged sixty-one, after suffering from prickling in the toes for two weeks, was suddenly taken with weakness in the left arm and right leg, and, a few days later, by the same paresis in the left leg. Within five days he could not stand or walk. The paralyzed feet hung flaccid, and were totally paralyzed; the thighs could be moved freely. Anæsthesia was found on the right sole and calf, and on the left foot and outer side of the leg. In the muscles of the hands and forearms a considerable atrophy, with paralysis, developed. The faradic contractility was abolished in the paralyzed muscles. He complained of a painful numbness in the paralyzed limbs up to the knees, and involving the hands. No improvement; death in four and a half months.

Autopsy: Spinal cord and nerve-roots were normal. The finer nerve-branches in the legs and hands were degenerated, only a small number of nerve-fibres being found. Single fibres showed no continuous myelin sheath; but this was segmented and granular. There was an increase of connective tissue, and many fat-cells in the nerves.[1]

This case being of an anomalous character excited very little attention. Two years later, however, Dumenil reported another, and published an elaborate article on peripheral paralysis, in which he said: "My own observations have convinced me firmly that many paralyses of obscure origin are caused by true spontaneous neuritis."[2] The second case, which with the first led to this conclusion, has been cited by Leyden as a case of neuritis, but it does not conform to any of the types of multiple neuritis, and is open to some doubt from the results of the autopsy, since numerous foci of disease were found in the spinal cord. We therefore pass it by.

---

[1] Dumenil: Gaz. Heb., 1864, p. 203, and Gaz. Heb., 1866, No. 4.

[2] Ceque j'ai observé me donne la conviction intime que bien des paralysies de cause obscure ont leur point de départ dans les véritables névrites spontanées, Gaz. Heb., 1866, No. 4.

Singular as it may seem, an interval of ten years elapsed before another case of similar nature, accompanied by a record of *post-mortem* examination, appeared. Then Eichhorst, of Berlin, reported the following interesting history :

Observation II.—A female, aged sixty-six, after suffering daily for two weeks from a chill, fever, and sweat, attended by malaise, anorexia, and constipation, noticed an œdematous swelling of both feet and legs, and complained of pain in the abdomen. A week later, on admission to the hospital, these symptoms continued. The urinary examination was negative. Three days after admission she suddenly felt a severe boring pain in the left leg, shooting into the toes, and at the same time a profuse sweat broke out over the calf and back of the foot. A few hours later a total paralysis was found in the muscles supplied by the peroneal nerve, with a marked anæsthesia. The electric reaction, at first preserved, was found two days later to be gone. After six days, during which she had no further chills, a paralysis of the anterior tibial nerve developed, and soon after of the posterior tibial nerve also. One week later entire paraplegia of the legs, with anæsthesia, severe pains, continual sweating, increasing œdema, and loss of the tendon reflexes had ensued. There followed a paralysis of the left, and soon after of the right, radial nerve. Moderate fever continued, and albuminuria appeared. Three weeks after her admission sudden blindness developed, the ophthalmoscopic appearances being at the time normal ; the patient then lay in bed with eyes closed, unable to move a limb. The extremities perspired constantly, and were tender, any pressure on nerve-trunks being very painful. In the face there was no trouble, and the senses were normal except that of sight. No trouble in swallowing. Nothing abnormal about the viscera during the entire disease. No irregularity of pulse or respiration. Death occurred on the forty-fourth day of the disease.

Autopsy : Spinal cord absolutely normal. The nerve-trunks in the bicipital grooves appeared intensely red to the eye, the perineurium being discolored and the endoneurium blood red. The same appearance was noticed in the large nerve-trunks of the arm in their course, as well as in the left tibial nerve. The microscopic examination showed a remarkable distention and tortuosity of the blood-vessels of the perineurium ; the vessel-walls were thickened, their nuclei increased. In the vicinity of the vessels a large number of lymphoid cells were found, which everywhere followed the vessels and infiltrated the connective tissue. There were also numerous fatty cells. The connective-tissue fibrillæ of the perineurium were thickened, shining, and swollen; their nuclei were increased and partly infiltrated with fat granules. Similar changes were seen in the endoneurium, viz., numerous extravasations of blood, which separated the nerve-fibres and compressed them. The nerve-fibres showed marked degeneration, especially those lying next the endoneurium, consisting of disintegration of the myelin sheath, and a distention and spindle-shaped swelling of the individual nerve-fibres. The nuclei of the sheath of Schwann were not increased in number, but the protoplasm about them was coarsely granular and opaque. The cells of the endoneurium were everywhere wanting, being replaced by fatty granular cells, even between the uninjured fibres.[1]

This is the first case to be found in which the microscopic appearances are described with sufficient detail to be satisfactory. It is to be noticed that here the lesion was an acute inflammation, and was, apparently, a diffuse one, both interstitial tissue and nerve-fibrils being involved in the process. That it was primarily an interstitial inflammation, and that the affection of the nerve-fibrils was secondary, due to pressure of the products of exudation, is clearly seen in the fact that those fibres were more seriously affected which lay near vessels, while the deeper fibres in large bundles were not at all degenerated. The appearance of the fibres was such as occurs in any degeneration from pressure. There is no reason, therefore, to believe that the process began diffusely.

As to the symptoms in this case, it is to be remarked that they were ushered in by an acute febrile movement with chills, and that severe pain was an early and prominent symptom; also that œdema and sweating were present; and that the optic nerves were involved.

The clinical picture in the following case of Joffroy, published three years later, was somewhat different, as was also the pathological condition.

Observation III.—A washerwoman, aged thirty-three, in the last stage of phthisis, was admitted to the hospital on March 5th. In February she had noticed a rapidly increasing weakness of her legs, and at the time of admission she could not walk or raise her feet from the bed. She could flex the knees but not extend them. There was no contracture, and the muscles were relaxed and flabby. Sensations to pain, temperature, and pressure impressions were normal, but the muscular sense was lost, and reflexes were diminished. She showed such a degree of mental weakness that tactile sense could not be tested. She had no shooting pains, no loss of control over bladder and rectum, no bedsores. Two weeks later the arms became involved in the paralysis, and atrophy, inco-ordination, and loss of muscular sense, with fibrillary motions, developed within a few days. There was great diminution of faradic excitability in all the paralyzed muscles. At the end of ten days the arms were entirely powerless, but retained their sensibility. On April 7th she died.

---

[1] Eichhorst: Virchow's Archiv, Bd. 69, p. 265. 1876.

Autopsy: A chronic meningitis of the brain explained the mental symptoms. The spinal cord was normal. The nerves appeared normal; but microscopic examination showed very marked degeneration in all the nerve-trunks, but especially in the sciatic, radial, and ulnar nerves. There was a segmentation of the myelin sheath, which at places was reduced to a finely granular mass. Many sheaths of Schwann were filled with this mass, others were empty. The nuclei of the sheaths of Schwann were increased in number. All the spinal nerve-roots were normal. The changes in the nerves were followed down into the fine terminal branches in the thenar muscles. The muscles were atrophied and showed fatty degeneration.[1]

Here, in contrast with the preceding case, it is to be noted that the lesion was not attended by congestion of the nerves, or by any exudation of lymphoid cells, or by marked interstitial changes. The affection was a true parenchymatous inflammation, with degeneration of the myelin sheath and axis-cylinder. As a result, the macroscopic appearance of the nerves was not such as to attract attention, and it required a microscopic examination to demonstrate the changes present. Joffroy, who reports this case as one of general spontaneous neuritis, finds the lesion identical with that observed in cases of localized neuritis occurring from cold, from lead palsy, or as a sequel of the infectious diseases. In regard to the symptoms, also, the case contrasts strongly with the preceding one. The patient had phthisis. The disease advanced more slowly. Pain was absent, and the sensory symptoms were by no means prominent, the muscular sense being the only one affected.

In 1880, the following cases were observed by Leyden, in the Charité Hospital of Berlin, in both of which changes were found in the peripheral nerves. They are cited because they not only enlarge our clinical picture, but confirm the pathological conditions already described.

Observation IV.—A sailor, aged twenty-five, was suddenly seized with severe tearing pains in all four extremities, especially near elbows and knees, which were swollen, white, indurated, and very tender. The pains shot down the limbs, and were attended by formication and by diminished sensation in fingers and toes. High fever accompanied the onset. In a few days, as the fever subsided, a paretic condition with muscular atrophy was noticed in the forearms, and to a less extent in legs and feet. A gradual improvement took place in the course of a few months in the lower extremities, but the paralysis and atrophy in the arms increased in degree, and the appearance of the hands resembled that seen in lead palsy. There was a reaction of degeneration in the muscles paralyzed. A year after the onset he died of chronic nephritis.

---

[1] A. Joffroy: Arch. de Phys., norm. et path., 1879, pp. 172–198

Autopsy : A sclerotic atrophy of both radial nerves was found. The nerve-sheath was thickened. The muscles were considerably atrophied. The anterior spinal nerve-roots, as well as the cells in the anterior horns, and the entire spinal cord, were normal. In the nerves of the lower extremities no changes were visible.[1]

Observation V.—The second case was that of a merchant, aged thirty-one, who noticed about Christmas, 1878, a feeling of formication in the toes and sole, of first the left, and then the right foot. This continued more or less severe, without any motor disturbance, until July 10th, when he found his legs heavy, and in two days this had so increased that he could not walk. At the same time a constant formication in the soles of the feet was felt. Within a few days similar symptoms appeared in the arms and hands. On admission to the hospital on July 17th, he complained of weakness of the legs and loss of sensation in the hands, also burning pain in the left hand and inner side of the left knee. The motion of the legs was powerless and uncertain, and there was paresis of the extensors of the hands and fingers. Sensation was much impaired in the periphery of the extremities ; the tendon reflexes were abolished, the muscles flabby. The electrical examination showed a loss of faradic contractility, and a great diminution of galvanic contractility in the affected muscles. The liver was tender, and there was a slight jaundice ; pulse 70 ; no fever. The paralysis increased slowly in extent and severity, and was accompanied by paræsthesia, pains, and diminished sensibility. The muscles atrophied, and showed the reaction of degeneration. After six weeks slight fever began, with rapid pulse aud dyspnœa. The pains affected the upper part of the thorax, and the spinal column became sensitive as low as the eighth dorsal vertebræ. During the last days small rapid pulse, severe dyspnœa, purely costal respiration, delirium, somnolence, and exhaustion were noticed, and on September 3d he died.

---

[1] Leyden : Charité Annalen, 1880.

Autopsy: The spinal cord was normal, the gray and white substance intact, the large ganglion-cells in normal form and number, though a few seemed to present a slightly swollen, glassy appearance which Leyden considered the first indications of trophic disturbance. The nerve-trunks, especially the radials and peronei, presented the appearance of a high degree of fatty degeneration and atrophy. The myelin sheath was either wholly wanting or greatly divided and split up, and in a state of fatty degeneration; the axis-cylinder seemed varicose from deposits of fat in it. A marked infiltration of lymphoid cells, especially between the endoneurium and nerve-fibres, was noticed, more marked along the course of the small arterioles. These changes were present in a greater degree in the peripheral branches of the nerves, and seemed less in the nerves near the cord.[1]

It will be noticed that here the lesion corresponded quite closely to that in the case of Eichhorst.

Impressed by the striking features of these two cases, and by their similarity, both in clinical symptoms and pathological changes, to the cases of Dumenil, Joffroy, and Eichhorst already cited, Leyden undertook a review of atrophic paralysis.[2] He showed the confusion which had come from blindly referring all such cases to a single lesion in the spinal cord. He proved that previous authors had erroneously grouped together several diseases distinct from one another, both in their course and in their pathology. One of these is poliomyelitis anterior, acute and chronic, occurring in children and adults; a disease of the anterior gray horns, with local foci of inflammatory degeneration; a disease characterized by motor and trophic symptoms of peculiar distribution and typical course. A second of these is progressive muscular atrophy, with its corresponding bulbar affection, glosso-labio-laryngeal paralysis; a disease of the anterior gray horns and of the motor cranial nerve-nuclei, of a chronic degenerative, non-inflammatory kind; a disease characterized by trophic and motor symptoms of different distribution, nature, and progress from the first disease considered. A third disease is pseudo-hypertrophic paralysis, in which a fatty deposit in the muscle conceals the real atrophy in progress; a disease of childhood, but not due to any spinal affection;

---

[1] Leyden: Zeitsch. f. Klin. Med., 1880.

[2] Leyden: Zeitschrift für Klin. Med., I. Ueber Poliomyelitis und Neuritis.

a disease of the muscular tissue itself. And lastly, he drew the picture of multiple neuritis, a disease due to degenerative processes in the nerves, independent of spinal lesion; a disease characterized by atrophic paralysis, associated with marked sensory symptoms, and with tenderness of the nerve, by a typical course, and, usually, by a favorable termination. By this article Leyden established the status of multiple neuritis as a distinct disease. Its symptoms were analyzed. Its lesion was described. The pathological stage in the progress of its discovery was complete.

When the characteristic features of a new disease have once been clearly pointed out, it is remarkable to observe how rapidly cases of it begin to be recognized. In the two or three years which followed the appearance of Leyden's article, numerous cases of multiple neuritis were reported in the journals. And many physicians, reviewing their records, recognized, in cases previously obscure or imperfectly diagnosticated, typical pictures of the new disease. It may be well to consider a few of these cases in order to complete our clinical knowledge of the affection.

Observation VI.—A female, aged thirty, of intemperate habits, but otherwise in good health, after suffering from formication, coldness, and pains in her feet and legs for some months, noticed an œdema of both legs. This increased rapidly after a few days, and the swollen limbs became painful to touch or pressure, and were the seat of severe lancinating pains, which were worse at night. Within a month the same symptoms appeared in the arms and hands, and a marked hyperæsthesia developed in all the extremities, as well as a rapidly progressing paralysis; so that, on admission to the hospital, six weeks after the appearance of the œdema, it was impossible for her to lift her limbs from the bed, or to extend her hands and fingers. The movements in the distal portions of all the extremities were much more impaired than those near the trunk, and in the paralyzed extensor muscles the faradic excitability was almost abolished. Heart and kidneys normal. She had some fever, and was delirious at night. The symptoms increased rapidly; the paralysis became total; respiration became difficult; the heart rapid, and a week after admission to the hospital she died.

Autopsy: Tubercles in the lungs. Brain and cord were normal. Nerve-roots were normal. In the nerves of the extremities marked degeneration was found, especially in the radial and tibial nerves. By the side of a small number of empty sheaths were found fibres, whose myelin was segmented and in drops, separated by empty spaces. The axis-cylinders were indistinct. There was no increase in the nuclei of the sheath of Schwann.

In discussing the case Dr. Lancereaux made the diagnosis of alcoholic paralysis, assigning the lesion to the nerves, and differentiating it from a myelitis or a meningitis. In so doing he criticised the view of Wilks and Lockhardt Clarke, who still considered alcoholic paralysis as a central disease. He cited another case, very similar, of a female aged thirty-three, in which the symptoms were pains, hyperæsthesia, tenderness, paralysis, with atrophy in the extremities, but in which œdema was not so marked and came on quite late in the course of the case. The same lesions were found. To these he added two cases in which the patients manifested the same symptoms, but had never drank. They were both, however, sellers of varnish, and lived day and night in an atmosphere permeated by alcoholic vapor, from which he concludes that chronic alcoholic poisoning can occur by absorption through the lungs—a valuable observation, but hitherto not confirmed.[1]

The following case was reported by Grainger Stewart, together with two other cases which resulted in recovery.

Observation VII.—A male, aged thirty-one, noticed during August, 1880, a weakness of the legs, and during the following month a pain of a prickling character in the legs and feet. These increased in intensity, and in October a similar feeling came on in the fingers and hands, accompanied by loss of power and stiffness. When seen, in November, he had tingling pain in both legs from the knee to the back of the foot, with numbness and feeling of cold in the toes and plantar surfaces, so also in the hands, to a less extent. No girdle pain or formication. Sensibility to touch was diminished in the legs and hands. Transmission of impressions was delayed. Sensibility to heat, tickling, and pain were all diminished, as was also the muscular sense in the feet. There was no nystagmus, although he complained of things dancing before his eyes. Sight was normal. There was no incontinence of urine or fæces. The skin reflexes were absent in the soles, but normal in the abdomen and groins. The knee-jerk was absent. Voluntary motion was greatly impaired in legs and arms, and attempts to use the muscles caused pain. Electric irritability of the muscles and sensibility of the skin was much diminished. There were no vaso-motor or trophic changes. His mental condition became changed during his stay, his memory was impaired, and he seemed drowsy. A month after his first examination he died of pneumonia.

---

[1] E. Lancereaux: De la Paralysie alcoolique, Gaz. Heb. de Méd., 1881, p. 120.

Autopsy by Dr. D. J. Hamilton: The median, ulnar, and tibial nerves showed great changes. With a low power of the microscope the bundles of fibres appeared to be affected by fatty degeneration. With a high power it was found that the axis-cylinders were swollen so as to form a number of fusiform bodies in the course of the nerve-tubes. These at parts were divided into a number of round homogeneous colloid bodies. When set free these bodies underwent a fatty degeneration, forming compound granular corpuscles. In some fibres the axis-cylinder was totally destroyed, nothing but a quantity of fibrous tissue remaining. The cords of the brachial plexus and the sciatic nerves were normal. Slight evidence of secondary sclerosis in the spinal cord was found in the columns of Goll and in the direct cerebellar columns. Its origin could not be explained.[1]

It must not be supposed, from the fact that all the cases so far cited were fatal, that death is always the result in multiple neuritis. This is very far from the truth, and probably, if the mortality had been greater, the pathology of the disease would not so long have eluded search. The fatal cases have been brought together in order that the pathological appearances observed might be noted and compared, and might become somewhat familiar by repetition. It is time to enter upon the more careful study of their pathology, and so, for a time, let us leave the clinical features of the disease.

It may not be out of place, before proceeding to discuss the changes occurring in inflammations of the nerves, to review the normal anatomy of a nerve.

THE HISTOLOGY OF A NERVE-FIBRE.—When a nerve-trunk is dissected, the connective-tissue sheath or perineurium enclosing its fibres torn away, and the individual fibres set free by teasing from the finer connective-tissue strands or endoneurium which bind them together, it is possible to distinguish certain parts, by means of appropriate methods of staining.

There is *first* the axis-cylinder, Fig. 1 (*a*). This is made up of a number of primitive fibrils arranged longitudinally and continuous throughout the length of the nerve. The fibrils are cemented together by a substance which appears finely granular. Each cylinder is supposed to represent a prolongation of a single nerve-cell; but whether the individual fibrils of which it is made up come from the same cell or from different cells, or from the fibrillary network in the central organ surrounding the cell, is a matter of hypothesis.[1]

---

[1] Grainger Stewart: Edinburgh Medical Journal, April, 1881.

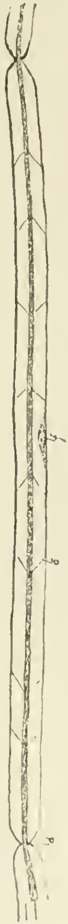

FIG. 1.—A Normal Nerve-fibre. *a*, Axis-cylinder; *b*, medullary sheath; *c*, sheath of Schwann; *d*, node of Ranvier; *e*, incisure of Schmidt; *f*, nucleus of the sheath of Schwann

There is, *secondly*, the myelin sheath surrounding the axis-cylinder, Fig. 1 (*b*). This is not a continuous tube, but consists of a series of little tubes, or interannular segments, placed end to end. The point of junction of two adjacent segments is indicated by a constriction in the contour of the nerve-fibre, and if the myelin be stained with osmic acid it is seen to be deficient at these constrictions. If, by any means, the nerve-fibre be broken, the myelin in any segment will run out and collect in drops, showing that it is a semi-fluid substance, of fatty nature.[2]

There is, *thirdly*, a connective-tissue membrane surrounding the myelin sheath, the sheath of Schwann, Fig. 1 (*c*). This, like the last, is made up of segments cemented to one another at the point of constriction of the fibre. This constriction is known as the ring or node of Ranvier, Fig. 1 (*d*), and at its situation the sheath of Schwann is the only covering of the axis-cylinder. On the inner side of this sheath, and half-way between two nodes, is found a nucleus, Fig. 1 (*f*). The sheath and node are made evident by staining with nitrate of silver, while the nucleus is seen best by staining with carmine or acid fuchsin. The mutual relation of the sheath of Schwann and the myelin sheath is best understood by the study of their development, segment by segment. Ranvier[3]

---

[1] Whatever their origin, it is certain that they pass together in the axis-cylinder to the periphery, and that there the axis-cylinder as such terminates, while the individual fibrils branch out in various directions, and, joining with other fibrils from other cylinders, form a fine plexus within the organ to which the nerve as a whole has gone. It has been thought possible to trace individual fibrils of the plexus into individual epithelial cells. Such is their termination in various internal organs and in the skin. This is not, however, the only manner of termination of the axis-cylinder, for individual nerves can be traced directly to terminal organs, such as the terminal plates upon the muscle, and the terminal bulbs and corpuscles in the skin; in which cases no division or branching of the fibrillary constituents of the axis-cylinder has been discovered. The termination in each of the organs of special sense is still different. The axis cylinder can best be seen by the acid fuchsin stain.

[2] The myelin sheath is not a necessary constituent of all nerve-fibres, for the majority of the nerves of the sympathetic system are devoid of such sheaths. Nor does the myelin sheath of the nerves of the cerebro-spinal system extend from end to end of the axis-cylinder. For the axis-cylinder first receives its sheath at some little distance from the cell from which it issues, and at its termination, where it breaks up into branching fibrils, the myelin envelope ceases. Throughout the course of the nerve, however, the myelin sheath is present in the cerebro-spinal nerves, forming a protecting envelope, and probably acting as an insulating substance as well.

[3] Ranvier: Leçons sur l'Histologie du Système Nerveux, tome i., p. 115.

likens the production of any one segment of these sheaths to the production of a fat cell from a nucleated cell. Every nucleated cell is surrounded by a layer of protoplasm. As the fat forms it collects within the layer of protoplasm in the vicinity of the nucleus, in the form of drops which finally unite into a mass. This mass is surrounded by a very thin layer of protoplasm and by the cell-membrane. On one side of it is seen the nucleus flattened out against the inner side of the membrane in the layer of protoplasm. Now, if each segment of the sheaths corresponds to the fat cell, and the myelin to the fat, the structure of the segment is clear. The membrane of the fat cell is represented by the sheath of Schwann. At the point of constriction of the nerve-fibre this membrane is cemented to that of the adjoining segment. Beneath the membrane, and flattened against its side, is the nucleus of the segment, lying in a layer of protoplasm in the middle of the segment. This layer of protoplasm forms a lining everywhere to the sheath of Schwann. With that sheath it is reflected around the axis-cylinder. Within the lining, in a state of semi-fluidity, is contained the myelin, which will run out if the limiting membrane is broken. By appropriate staining fine lines can be seen passing between the outer and inner layers of protoplasm, through the myelin sheath, the so-called incisions of Schmidt, Fig. 1 (*e*). These have been considered little trabeculæ of protoplasm within the segment in the midst of which the myelin lies. Recent staining methods seem to indicate that they belong to the sheath of Schwann and are connective tissue. The layer of protoplasm lying against the axis-cylinder is the layer which was formerly described as the sheath of Mauthner. Some authorities consider that a layer of connective tissue similar to the sheath of Schwann surrounds the axis-cylinder, but this is still uncertain. Thus the nerve-fibre consists of a central conducting strand, surrounded and insulated by a series of tube-like segments of fluid contained within a membrane, which are joined to one another, forming a double protecting sheath.

Individual fibres are associated in bundles held together by fine connective-tissue cells whose nuclei can be seen in a carmine or fuchsin stained preparation, lying always adjacent to, but outside of, the sheath of Schwann.[1] This has been called the endoneurium, while the connective-tissue sheath surrounding the entire bundle is named the perineurium. Capillary vessels with free anastomoses run within the nerve, their walls lying adjacent to the individual fibres, and thus affording a perfect nutrition.[1] Lymph-spaces also have been demonstrated within the nerve-sheath, but not among the fibres.[2] That the interfibrillary spaces of the endoneurium, however, open into these peri-fascicular lymph-spaces is probable, from the fact that they do so in other organs. While it is evident that the nutrition of the axis-cylinder is derived from the circulatory fluids, it is probable that it is only at the nodes of Ranvier that the absorption takes place, since elsewhere the myelin sheath interferes with osmosis. Thus, if the nerve be put in nitrate of silver, it is only opposite the nodes that the axis-cylinder becomes stained.

---

[1] These connective-tissue cells which lie between individual fibres have been recently the subject of study by Adamkiewicz (Adamkiewicz, Ueber Ruckenmarkschwindsucht, Sitzungsbericht der Wiener Acad., Bd. 91, Abth., II.) and Rosenheim (Rosenheim, Arch. für Psych., xvii., p. 830). The former regarded them as nerve-corpuscles, but the latter has proven that they are true connective-tissue cells. They are best seen in specimens stained with a double staining of anilin gentian violet and methyl blue, though they can be seen distinctly in fuchsin stained preparations, as has recently been demonstrated by Dr. Van

THE PATHOLOGY OF MULTIPLE NEURITIS.—In studying the pathological processes which occur in multiple neuritis, it is necessary to keep these various elements of the normal nerve in mind, since each element is subject to changes. The exact character of these changes is best understood by observing the results of nerve-degeneration artificially produced in animals. And a study of this will not be out of place here, for, as we shall see, the changes occurring in multiple neuritis correspond quite exactly to those produced by experimental degeneration.

In considering the pathology of degenerative neuritis we enter at once upon a mass of controversial statements. It would seem to be a simple matter to establish, by observations upon nerves which had been experimentally compressed or severed, the changes which ensue in nerve injuries. But, as a matter of fact, there is, perhaps, no field of experimental pathological inquiry in which the results have differed more widely. In the first place, it is probable that the rapidity and even the character of the changes differ in different animals. Secondly, various methods of investigation, of hardening, dissecting, and staining the nerves, seem to have resulted in the production of different appearances. And, lastly, it is by no means certain that a uniform pathological process goes on after experimental lesions.

After a lesion of a nerve-trunk, a process of degeneration sets in at the point of injury, and involves a small portion of the central end and the entire peripheral part of the nerve, from the seat of injury onward. This pro-

---

Giesen, of the Laboratory of the College of Physicians and Surgeons. They appear on nerves after the age of five, and increase in number as the individual grows older. so that in elderly persons they are very numerous. They are round or spindle-shaped, and frequently appear to have little thorn-like processes. In addition to these connective tissue cells there are fine fibrils of connective tissue, and bands of tissue lying between the bundles of nerve-fibres.

[1] Ranvier, l.c., p. 253.

[2] Ranvier, l.c., p 258.

cess may be more or less complete, and may, or may not, be followed by a second process of regeneration in the diseased nerve. It is necessary to distinguish between the degenerative and regenerative processes ; and, inasmuch as it is affirmed that they may proceed simultaneously in various parts of the same fibre,[1] it is not strange that the confounding of the two should have increased the confusion in the statements.

In early times it seems to have been a question whether the destruction of a nerve was ever recovered from, and while the majority of authorities rightly contended that this was possible, there were those who denied it vehemently. Steinneck,[2] in 1838, seems to have determined this finally in the affirmative, using both the physiological and anatomical data at his disposal to good advantage. The exact processes of degeneration were first carefully studied by Nasse[3] and Waller,[4] and their results having attracted attention to the phenomena in question, a host of investigators have followed their lead. The most careful examinations of the entire series of changes which go on in degeneration and regeneration have been made by Ranvier,[5] Philipeau and Vulpian,[6] and Dejerine,[7] in France ; by E. Neumann,[8] S. Mayer,[9] and Wolberg,[10] in Germany ; by Hanken[11] in Holland ; by Schiff[12] and Tizzoni,[13] in Italy, and by Weir Mitchell[14] in America.

The majority of writers upon nervous diseases and upon general pathology seem to have followed Ranvier closely, without any mention of the fact that other authorities differ from his conclusions. It will be necessary here to present the various views which are held.

THE PROCESS OF DEGENERATION.—When a nerve is compressed by a ligature or forceps without sufficient force to rupture the sheath of Schwann, the myelin is driven away from the point of pressure in both directions, and the axis cylinder is disintegrated and mingled with it. It might be supposed that the nodes of Ranvier would prevent such a driving back of the myelin, but they seem to offer but feeble resistance, so that the entire fibre on either side of the compressed spot is bulged out for some little distance, the sheath of Schwann between the distended portions being left empty or containing only a little granular débris. In a short time changes of a degenerative character are observed on both sides of the point of compression. Those on the central side are limited to the immediate neighborhood of the injured spot, and, according to Ranvier, do not affect the nerve for a distance greater than a centimetre from the point of compression.[1]

---

[1] E. Neumann : Ueber De- und Regeneration der Nerven, Arch. f. mikros. Anat., xviii.

[2] Schmidt's Jahrbuch., Bd. 26, S. 102, 1840.

[3] Müller's Archiv, 1839.

[4] Comptes rendus, 1852, vol. 34, p. 675.

[5] Leçons sur l'Histologie du Système Nerveux.

[6] Gaz. Méd. de Paris, 1859, Comptes rendus à la Soc. de Biol.

[7] Arch. de Physiol., 1875, p. 567.

[8] Arch. f. mikros. Anat., xviii., 302, 1880.

[9] Zeitsch. f. Heilkunde, ii., 1881.

[10] Deut. Zeitsch. f. Chirurgie, xviii. u. xix., 1883.

[11] Internat. Monatsch. f. Anat. und Histologie, Bd. iii., 1886 ; Fortschritte der Medicin. 1886, December.

[12] Comptes rendus, 1854, vol xxxviii., p. 448, and Zeitsch. f. Wissen. Zool., 1856, vii., p. 145.

[13] Centralbl. für Med. Wissen., 1878, Nr. 13.

[14] Injuries of Nerves, 1872.

On the peripheral side of the point of pressure the degeneration is extensive, involving the entire nerve down to its finest terminations. The process is held by some to be a gradually advancing one along the fibre from the point of pressure outward, while others believe that it begins at once in the entire length of the nerve. The first change noticed is a breaking up of the myelin sheath into segments, and then into smaller masses and drops (Fig. 2, *a*, *b*), which finally undergo further disintegration, either by a fatty or albuminoid degeneration, or by a process of saponification, until a finely granular mass alone remains. Tizzoni states that this process is partly due to the activity of migratory white blood-globules, a view which Ranvier supports, and which Neumann and Mayer deny. If, as seems probable, the connective-tissue cells of Rosenheim take an active part in all processes of nerve inflammation, it is possible that these are the bodies which Tizzoni mistook for leucocytes. Ranvier holds that the segmentation of the myelin is due to the increase of the protoplasm about the nucleus of the segment, and that it is this protoplasm which replaces the myelin. Neumann holds that the granular mass resulting from the disintegration is not protoplasmic, but is a débris capable of acting as the basis for processes of regeneration after undergoing a chemical change. All authorities admit that the granular mass may be gradually absorbed, leaving the sheath of Schwann collapsed and empty, or containing only granules of débris or nuclei (Fig. 2, *c*, *d*).

---

[1] Some recent investigations of Friedlander and Krause throw doubt upon this statement. (Fortschritte der Medicin, December 1, 1886.) They have discovered a considerable degree of atrophy in the nerve-trunks in the stumps of amputated extremities, and they traced this atrophy upward through the nerves, and into the posterior nerve-roots exclusively. They conclude, therefore, that the fibres affected are entirely sensory fibres. The axis-cylinder is only slightly affected by the atrophy, but the myelin sheath is wholly destroyed. Inasmuch as the degree of atrophy in a nerve-trunk was about the same whether the amputation had been made near the body or near the end of the limb, they conclude that only certain of the sensory fibres degenerate upward. And as the tactile corpuscles and terminal bulbs are only found in the most distal parts of the extremities, it is suggested that only those sensory fibres degenerate which arise from these so called tactile bodies. If this is proven to be true by further observation, the tactile bodies will assume the rôle of trophic centres for the sensory nerves. And in this case anything which interrupts the connection between the tactile bodies and the posterior spinal ganglion will produce a centripetal degeneration in the nerve. Hence in neuritis from section or pressure, as well as in multiple neuritis, such secondary degeneration is to be expected and should be looked for.

As the myelin undergoes these changes the axis-cylinder usually becomes involved. Some authors, it is true, believe that it remains intact, and that, although deprived of its function, it is capable of resuming that function at any time when regeneration of the myelin sheath has

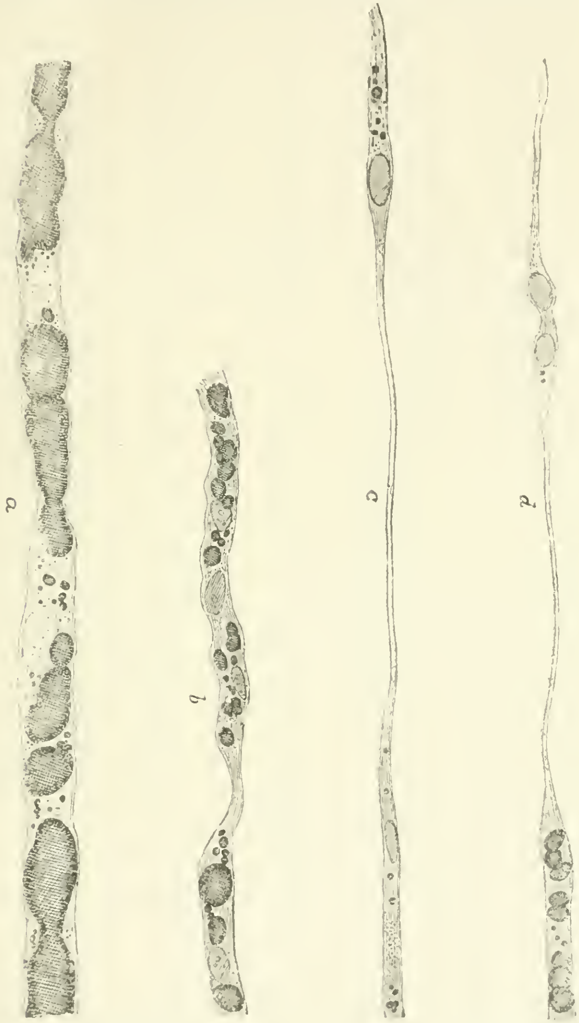

Fig. 2.—The Process of Degeneration. (After Ranvier.) *a*, Segmentation of myelin and axis-cylinder. with increase of protoplasm ; *b*, disintegration of myelin into drops, increase of nuclei, partial absorption of débris; *c*, complete absorption of the disintegrated mass, leaving the Schwann sheath empty, or containing only débris and nuclei ; *d*, the same, nuclei alone remaining.

taken place. Such authorities as Erb, Charcot, Weir Mitchell, and Wolberg have given their approval of this view. Ranvier believes that the protoplasm of the nucleus attacks and destroys the axis-cylinder as well as the myelin. Neumann holds that it is split up into segments like the myelin, becomes mingled with it, and undergoes the same process of chemical change, or absorption; and Mayer agrees with this view (Fig. 3, *a*, *b*, *c*).

The sheath of Schwann also takes part in the process of degeneration. When that process has fairly begun numerous nuclei are observed lining this sheath in each interannular segment. They may have come by a process of segmentation of the original nucleus of the segment, as Ranvier holds. But Neumann and Mayer have shown that they appear as early at the extremities of the segment as they do in the vicinity of the nucleus, and they therefore conclude that they are a free formation originating in the granular or protoplasmic mass. Tizzoni thinks them emigrated corpuscles. The most recent view of all is that of Rosenheim, that they come from the connective-tissue cells along the sheath which divide and multiply, and show powers of emigration as soon as the process of degeneration begins. When the granular mass is absorbed these nuclei remain scattered along the sheath of Schwann, and it has been suggested that, when in an empty sheath a new axis-cylinder appears, it owes its existence to these nuclei, which arrange themselves in a line and develop into the new fibre (Wolberg). If no regeneration occurs they disappear gradually, and then the only relic of the former nerve-fibre is the empty, collapsed sheath of Schwann, which remains as a connective-tissue strand.

The increase of nuclei and connective-tissue fibrils in the endoneurium and perineurium which accompanies the process of degeneration, aids in the transformation of the nerve into a band of connective tissue.

The degeneration which affects the nerve is continued to the terminal plates upon the muscle, and these are changed into a mass of granules and finally absorbed, a connective-tissue plate being left.[1] Whether any changes occur in the sensory terminal organs, such as the tactile corpuscles or terminal bulbs, has never been ascertained. Those who believe that the individual axis cylinder fibrils terminate in the epithelium of the skin may cite the trophic changes which often occur on the surface as evidence that this covering of the body shares in the nerve-changes.

Authorities seem to agree that the same results follow a division of a nerve that are observed after its compression, with the difference that at the point of section the myelin runs out of the sheath of Schwann. The cut central end of the nerve becomes swollen into a bulbous extremity by a growth of connective tissue.

---

[1] Gessler: Die Motorishe Endplatte und ihre Bedeutung für die Periphere-Lähmung. Leipzig. 1885.

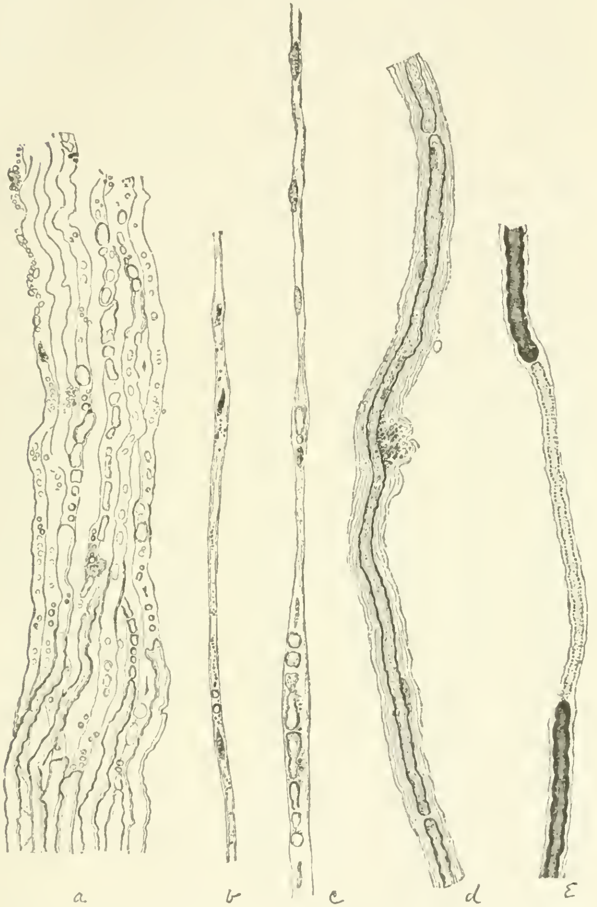

Fig. 3.—The Process of Degeneration and Regeneration going on in Normal Nerves. (After Mayer.) *a*, Segmentation of the myelin; *b*, contents of sheath of Schwann reduced to granular mass; *c*, sheath of Schwann, containing only débris and nuclei; *d*, granular mass in the sheath in process of regeneration; *e*, regeneration of a single segment after degeneration.

Whether a true union of the divided ends ever occurs is still a matter of uncertainty. The majority of authorities, following Ranvier, affirm that while a primary coaptation of the ends by an exudate which is secondarily transformed into connective tissue may occur and hold the ends in position, no true primary union of nerve-fibres is possible, and under all circumstances the degenerative process already described goes on to completion. Glück, however, claims to have observed an actual union of the two ends, with re-establishment of function, at a time too early to have admitted the occurrences of degeneration and regeneration ; and Wolberg, approaching the subject from the surgical side, and considering the results of nerve-suture, inclines to the same view.[1]

In the midst of such contrary statements, what conclusion can be reached as to the actual facts ? It seems evident that but one conclusion is certain, viz., that under different circumstances different processes occur. The various observers are equally trustworthy. It is impossible to choose one set of conclusions rather than another, or to rely wholly upon one series of experiments, however capable the observer may be, for they all rest upon repeated observations. It is undoubtedly true that in some cases the degenerative process, so graphically pictured by Ranvier, from the beginning segmentation of the myelin down to the final result in the connective-tissue strand, the relic of the empty sheath of Schwann, does go on ; while in other cases of a less serious nature the destruction is less complete, and there remains a fibre consisting of a sheath of Schwann, containing a granular mass which may be either an axis-cylinder or a mass capable of developing into an axis cylinder under favorable circumstances. If this is the case, we can affirm that brilliant surgical successes, with rapid restoration of nerve-function after suture, are possible when the partial degeneration is present, but are impossible when the total destruction of the nerve-fibre has occurred. And statistics show that there is a certain percentage of operations which fail of any result, though repeated on the same nerve.

It is evident that future research should be directed, not so much to determine which of the processes described occurs, as to settle under what circumstances the one is produced rather than the other. And such research will have an eminently practical bearing, inasmuch as it will also demonstrate under what circumstances nerve-suture is likely to be attended by success.

The Process of Regeneration.—After the process of degeneration has gone on for some time in the nerve-fibre, it may cease, and the process of regeneration may begin. With regard to the method of this process two widely divergent views are held. Ranvier and his fol-

---

[1] See, also, Weissenstein, Brun's Klin. Chirurg., 1884, p. 310.

lowers claim that the new nerve is wholly a product of the central end of the injured nerve, growing out from it and making its way along the track of the peripheral end, which takes no active part in the process. Neumann and Mayer, on the contrary, believe that the regeneration goes on in the peripheral end of the cut nerve, segment by segment being formed successively, beginning at the point of injury and proceeding outward, the entire nerve being built up by the union of each distal segment with the one lying centrally to it, until this process has reached the end. These various views demand a more exact statement.

Ranvier describes several ways in which the new fibres issue from the central end.[1] He has seen the central end of an individual nerve-fibre become hypertrophied, and from this swollen part a single new fibre start out already medullated. He has also seen a single axis-cylinder grow out, and then divide into two, or even more, axis-cylinders, each of which develops into a complete medullated nerve; or from the swollen central end of a single fibre several medullated and segmented fibres may originate, or one medullated fibre may be sent out with several non-medullated fibres coiled about it; or, lastly, a simple axis-cylinder branches out without a medullary sheath, divides into two, and each of these in turn divides into two, and so on until the sheath of Schwann, which had originally but one axis-cylinder within it, contains as many as eight or more (Fig. 4, *a*). These new fibres appear like a brush upon the end of the divided nerve, and wind about each other, forming a sort of plexus. By the aid of the cicatricial tissue, which usually joins the central end with the degenerated peripheral end, the bushy, branching fibres are directed outward toward the periphery. And when they reach the peripheral end of the cut nerve they insinuate themselves into the old remaining sheaths of Schwann, or between those sheaths, and grow on and outward till at last they reach the termination of the peripheral end, and the regeneration is complete. The terminal plates upon the muscles are renewed by a production of protoplasm in the plate. The description given by Neumann and Mayer is very different. It will be remembered that they describe, as the terminal result of the process of degeneration, a band of fibres each consisting of a sheath of Schwann containing a granular mass. The process of regeneration begins in this mass. Within it they have seen a narrow band of fine homogeneous substance appear, which has the structure of a rudimentary axis-cylinder (Fig. 3, *b*). This does not fill the sheath

---

[1] L. c., ii., 42–67.

of Schwann, and is often pressed aside by the nuclei which lie in that sheath (Fig. 4, *c*). It is not at first continuous with the end of the old axis-cylinder, remaining in the central part of the compressed or divided nerve, but as it increases in definite structure it approaches this old axis-cylinder, and finally unites with it. At the point of union a ring of Ranvier is formed. As this axis-cylinder develops, a substance is gradually formed around it, which is stained by osmic acid. This increases in thickness as the protoplasmic mass and the nuclei diminish, until it finally forms a new myelin sheath. The new myelin sheath is never continuous with the old one in the central end of the nerve, since it is separated from that by the ring of Ranvier; but often at first the older sheath seems to bulge out and encircle the new sheath, though this appearance is never permanent (Fig. 4, *c*). At the point of union of the new fibre with the old one, nuclei are often found; but these, like the others, gradually disappear (Fig. 4, *b* and *d*). Lastly, a new sheath of Schwann is produced around the new myelin sheath, and within the old sheath of Schwann. It presses aside the old sheath, together with such masses of protoplasm, drops of myelin, and nuclei as may remain, leaving them thus wholly outside of the new-made fibre, so that they coalesce with, and make part of, the endoneurium (Fig. 4, *e*). The new sheath of Schwann has but one nucleus in each segment, and presents the nodes of Ranvier at egular intervals.

Neumann has shown that this process goes on in every individual segment of the nerve-sheath, so that in segment by segment, proceeding toward the periphery, the regenerative change occurs, and as each segment approaches completion it joins itself to the preceding one (Fig. 4, *e* and *f*), until finally the entire nerve is re-established in its entire length. As the degenerative process begins in the segment nearest to the point of compression, so does the process of regeneration; and in some nerves the two processes may be seen going on together, the segments near the seat of injury being renewed while those at the periphery are still in a state of degeneration. The new fibres do not grow out from the old ones, as Ranvier describes in the regeneration of cut fibres, but protoplasm, with specific developmental properties, forms and differentiates the elements of the new fibre, and then unites it to the old one. The new fibres are, at first, somewhat smaller in calibre than the old ones (Fig. 4, *f*), but they gradually attain a normal size, and then the process may be said to be completed.

While Neumann would have the new fibres develop from the granular mass remaining in the old sheath of

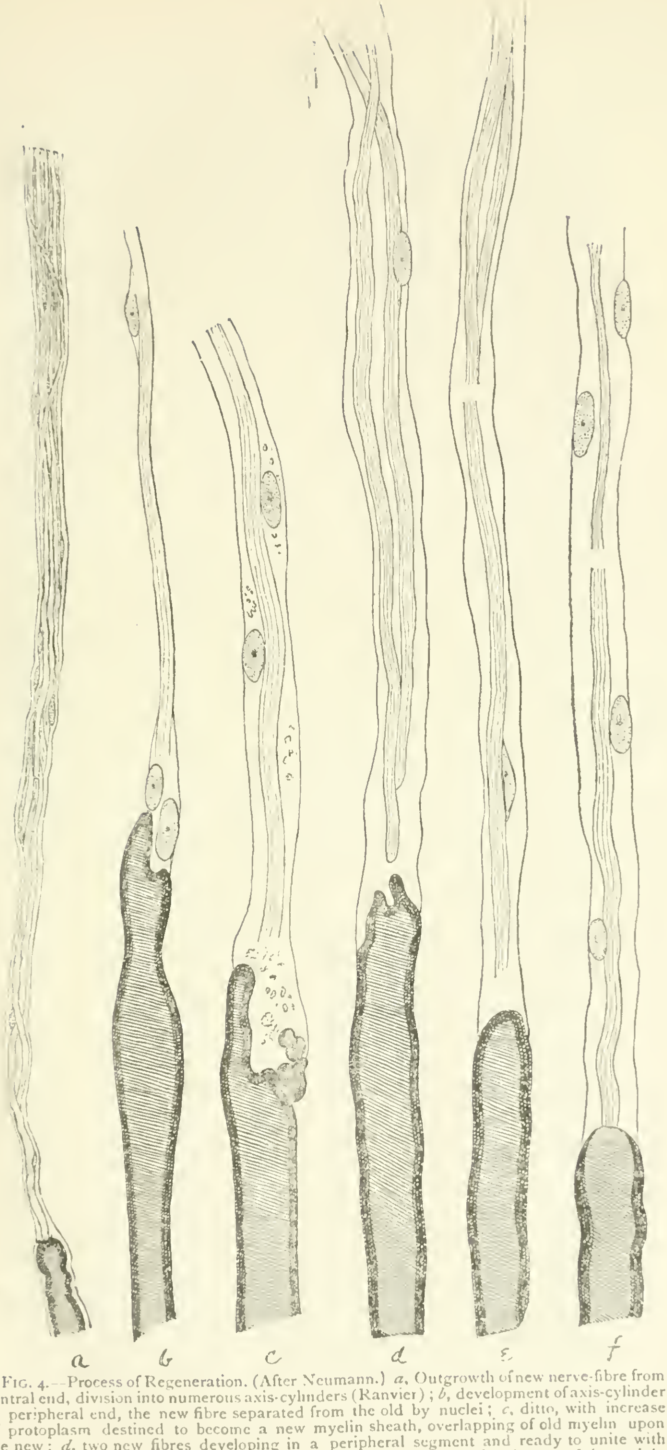

FIG. 4.—Process of Regeneration. (After Neumann.) *a*, Outgrowth of new nerve-fibre from central end, division into numerous axis-cylinders (Ranvier); *b*, development of axis-cylinder in peripheral end, the new fibre separated from the old by nuclei; *c*, ditto, with increase of protoplasm destined to become a new myelin sheath, overlapping of old myelin upon the new; *d*, two new fibres developing in a peripheral segment and ready to unite with the central end; *e*, two segments nearly developed, the central one in advance of the peripheral one; *f*, final union of the peripheral with the central segment, nuclei still remaining.

Schwann, Günther, Hjelt, and Weir Mitchell suppose that they may originate from the nuclei of the old sheath, or even from connective-tissue cells and neurilemma nuclei remaining in the connective-tissue strand, and to this opinion Wolberg agrees. It is evident, therefore, that the process of regeneration is by no means accurately determined. But here again it is possible that conflicting statements may be explained. The method of regeneration may, perhaps, depend upon the exact stage of degeneration reached before it begins. If the product of degeneration is a simple band of connective tissue, it seems probable that the nerve-fibre will have to grow into it from a central origin, as in its original development in fetal life, unless the connective-tissue cells recently discovered are neuro-plastic cells, and have the power of producing new nerves, just as cells of periosteum may produce a new bone. If, however, when degeneration ceases, there remains a sheath of Schwann containing a granular protoplasmic mass, it is not at all improbable that that mass may be differentiated into an axis-cylinder and a medullary sheath, and joined to the old nerve-fibre, a process which has its analogy in the medullation of nerves in the embryonal state. If we admit, with Wolberg, that in some cases the axis-cylinder is not destroyed, the formation of new myelin is a rapid matter. That some such process as the one last described must occur in certain cases is certain, when the rapid recovery after minor injuries is considered, and when the results of nerve suture are taken into account; for in both these conditions the return of function occurs long before a new nerve-fibre, starting out from the old one, could have reached the periphery.

It is affirmed by Mayer that individual nerve-fibres in normal nerves are constantly undergoing these processes of degeneration and regeneration, either because the necessary renewal of worn-out tissue takes place in this manner, or because slight injuries from pressure or over strain are sufficient to start up degeneration in single fibres.

The process of degeneration in the nerves, consequent upon the destruction of the ganglion cells from which they arise (the so-called Wallerian degeneration) differs in no respect from that ensuing upon compression or division, excepting that in the latter case only the distal part of the divided nerve undergoes the pathological change, while in the former it is the entire nerve that is affected. And in the changes described in multiple neuritis the same progress of events and the same varieties of termination are observed.

When the cases of multiple neuritis are examined from a pathological standpoint several varieties may be distinguished. In some of the cases, and these are the most numerous, there is a parenchymatous inflammation in the nerve-fibre only visible to the microscope. At the outset of this inflammation the myelin sheath appears slightly swollen, is less homogeneous, and, from a difference of refractive power, is less translucent. It then becomes split up into segments of different length and form, the segmentation occurring preferably at the incisures of Schmitt, while the incisures at other parts disappear. Between these segments of myelin a finely granular protoplasm is seen, in which new nuclei are found. These nuclei probably are the cells of Rosenheim which have emigrated, although a few near the nucleus of the sheath of Schwann may be due to its division. In some fibres the axis-cylinder may still be preserved. In others it is broken at the same places as the myelin. At the next stage of the process the changes are more marked. The myelin is now reduced to a series of small globules surrounded everywhere by granular protoplasm, and in this protoplasm the nuclei are now very numerous. The axis-cylinder cannot be distinguished in the mass, as a rule, but occasionally a fine line is seen passing through the mass, which may be a remaining cylinder. The succeeding stage presents a different picture. While up to this time the size of the nerve-fibre has remained about normal and uniform, it is now seen to vary. At places the fibre is still wide and filled with a granular mass, at other places it is narrow, the mass having disappeared, leaving either a collapsed sheath or a sheath containing only nuclei here and there. In a few such narrow fibres there seems to be an axis-cylinder lying directly within the sheath of Schwann, and occasionally separated from it at various places by nuclei. But this appearance is rarely seen. As a rule, no trace of the axis-cylinder remains. As any single fibre may show at some places constrictions, at others dilatations, the variation in its calibre is the most striking feature of this stage. In the terminal stage the calibre is uniform again, but is now everywhere reduced. The sheath of Schwann is empty, or contains only a little granular substance, and the nuclei are now less numerous than before. There is, in fact, only an atrophied tube with none of its original contents. These tubes, lying side by side, are folded and undulating, and appear like a strand of connective tissue.

These various stages of parenchymatous inflammation are to be seen in different fibres in the same specimen. Their appearance is identical with that observed in the course of degeneration of a nerve after compression, or after destruction of the spinal ganglion-cells. This has led such an accurate observer as Erb to advance the hypothesis that some slight changes in trophic cells in the spinal cord, not visible to the microscope, are present primarily, and that these changes are of a secondary nature.[1] But this cannot be admitted. Those who would explain the parenchymatous neuritis as secondary to changes in the cord, fail to explain why it should be limited, as it is, to the distal portions of the nerves, and not be continuous through the nerve-trunks and anterior nerve-roots into the anterior cells. They also offer no explanation for the cases in which the sensory, as well as the motor, nerves are affected, centrifugal degeneration of these nerves from spinal lesion being unknown. Strumpell, in meeting Erb's hypothesis, urges with reason that a parenchymatous neuritis has its parallel in other parenchymatous inflammations, and therefore does not need to be traced to any primary affection in trophic cells. He also mentions the existence of primary lateral sclerosis of the cord, similar in all respects to secondary degeneration of the pyramidal columns—yet now admitted to be a primary disease—as illustration of the fact that like changes are not always to be traced to one cause. It cannot but be admitted, therefore, that the first class of cases of neuritis must be considered as due to a primary parenchymatous inflammation in the nerve-fibres. This form seems to be much more frequent in its occurrence than the first variety, and constitutes the lesion in the majority of the cases hitherto reported. The appearance in section is seen in Fig. 5, *b*. The appearances of single fibres are the same as those shown in Figs. 2 and 3.

A second class of cases presents a different appearance. In this class the mere inspection shows the nerve to have been the seat of pathological changes, for it is either congested, swollen, and lacking in lustre, or it is yellow and irregularly swelled by the accumulation of fat, or it is evidently reduced to a mere connective-tissue strand. Upon teasing the nerve it is at once clear, from its brittleness, that individual fibres are lacking in continuity and are changed in structure. And if it is examined under the microscope the exudation of serum and of lymphoid bodies, the great increase in the number of connective-tissue nuclei, the distended condition of the vessels, as well as the various appearances characteristic of nerve degeneration, are clearly seen. Here the inflammation is either originally an interstitial inflammation or, more probably, a diffuse one. It is possible that the degenerative processes in the nerves may have been due to the compression by the exuded products of inflammation within the nerve-sheath. In one or two cases where the patient died early in the disease, the nerve-fibres which lay near the vessels were affected to a greater degree than those lying deeper, and from this fact it was concluded that their degeneration was secondary. In other cases, however, all the fibres in a bundle are equally involved. Here it is probable that the inflammation was diffuse from the outset, parenchyma and interstitial tissue being affected simultaneously. The final result in either case is a diffuse inflammation. One marked feature in these cases is the large amount of fatty deposit found in the altered nerves. This is to be ascribed to the fact that myelin in undergoing degeneration breaks down into globules and undergoes a fatty change. It is itself allied to fat, and making up, as it does, a large part of the bulk of the fibre, it would be noticeable in the product of degeneration if it were not absorbed. In this form the absorption seems to be interfered with by the vascular condition, and hence the residual amount of fat is increased. The changes observed in the nerve-fibres are very similar to those seen in the first form of neuritis, and need not be described again.

---

[1] Erb: Neurol. Centralbl., 1883, p. 481.

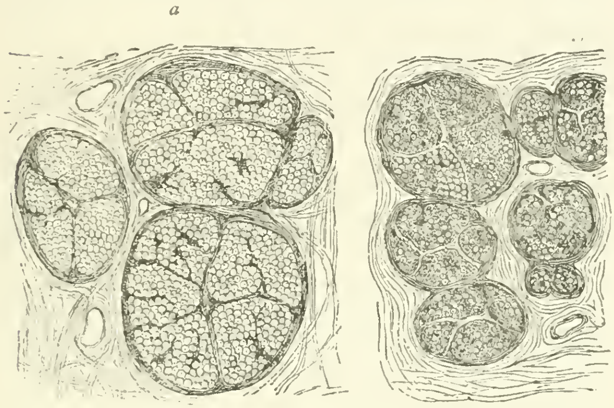

Fig. 5.—(After Joffroy.) *a*, Section through normal nerve; *b*, section through degenerated nerve in a case of multiple neuritis, bundles reduced in size, individual fibres degenerating, their place taken by débris.

It is to be noted that in both of these forms the pathological changes are always more intense in, and are occasionally limited to, the peripheral terminations of the nerves. The nerve-trunks may be slightly involved in their distal portions, but it is very rare to find any changes in them at their origin from the plexuses. And, as a rule, the spinal nerve-roots in cases of multiple neuritis are normal. In making autopsies upon such cases this fact is to be remembered, and the nerves are to be removed down to their finer branches in the muscles and fascia.

One additional pathological form must be mentioned, since it has been described by such a careful observer as Gombault.[1] It is the so-called segmental periaxillary neuritis. In toxic neuritis from lead-poisoning, Gombault found that the degenerative process was not uniform in the entire length of a nerve-fibre. On the contrary, entirely normal segments alternated with the degenerated segments in the nerve. Mayer has noticed a somewhat similar condition (Fig. 3, *e*). The changes already described take place in the myelin sheath of the affected segment, even to its entire absorption, leaving the axis-cylinder in the sheath of Schwann. But as the adjacent segments are not involved, regeneration is thought to be more easily accomplished. Pitres and Vaillard[2] have noticed a somewhat similar condition in the neuritis occurring after diphtheria, although in their case the axis-cylinder as well as the myelin sheath was totally destroyed in many segments.

While it is, of course, impossible to describe definitely the process which goes on to complete regeneration of nerves in multiple neuritis, there is no reason to suppose that it differs in any way from that observed in experimental lesions.

The time required for the completion of the process will depend upon the severity and extent of the degeneration. When that is slight the recovery may be rapid, cases having been reported where a total cure took place in two months. As a rule, however, it is a slow process. The large majority of the cases on record required over six months for the complete regeneration, and in not a few cases from ten to sixteen months elapsed before the condition of the nerves was proved to be normal by the total disappearance of all symptoms.

A degeneration of the muscles proceeds hand in hand with that of the nerves. This is usually in the form of an atrophy, with fatty degeneration and an increase of nuclei. The muscle-fibres become thin, homogeneous, are easily split up, contain fatty granules, and finally, in place of the atrophic muscle. new connective tissue is found. When no regeneration takes place, a mass of connective tissue replaces the muscle, containing a few muscular elements.

With the end of 1883 we may consider the third stage of pathological discovery in the history of multiple neuritis as terminating. At that date the symptoms of the disease had been recognized as constituting a distinct clinical picture ; hypothetical lesions in the spinal cord had been abandoned, and the exact pathology of the disease had been ascertained. Since 1883 the final period in the development of knowledge of the affection has been in progress. About one hundred cases, of varying degrees of severity, have been observed (see Bibliography). The lesions described have been confirmed in all their details. The symptoms arising in the course of the disease have been subjected to careful analysis, together with their varied modes of combination. Certain diseases hitherto considered of spinal origin are found to be due to peripheral disease. Thus it has been discovered that a form of ataxia may occur from multiple neuritis of alcoholic or arsenical poisoning which closely resembles and was formerly confounded with tabes ; also, that some cases supposed to be anterior poliomyelitis must now be differently named. And as physicians review their records they find that former diagnoses require revision in the light of new discoveries, and that greater circumspection is to be exercised in differentiating spinal from peripheral affections. The grounds for such differential diagnosis will require attention in the next lecture.

---

[1] Gombault: Archives de Phys., 1873, p. 592; also Arch. de Neurol., i., 1.

[2] Pitres et Vaillard: De la Névrite segmentaire, Arch. de Neurol., xi., 337.

And, what is perhaps of greater importance, the fact has been elicited that some combinations of symptoms, formerly supposed to be without a pathological basis, some of the so-called peripheral neuroses, really belong to this class of diseases. At present we have only time to allude to one or two forms of peripheral neuroses which must be removed from that unsatisfactory category of disease and be considered as peripheral neuritis.

And the first of these is the affection termed numb-fingers. This was first described by J. J. Putnam, of Boston, but met with instant recognition from neurologists and from general practitioners all over the world. It is a disease chiefly seen in women between the ages of forty and sixty, usually associated with dyspeptic or uterine symptoms, but entirely independent of them. It begins as a tingling sensation in the ends of the fingers, felt at night, and sufficiently annoying to keep the patient awake. It then extends to the entire fingers, and may invade the hand, and is felt by day as well as by night. The fingers are so numb that all finer acts become impossible ; the patient can no longer sew or knit, cannot be sure of holding anything securely, and finds herself unable to perform any delicate movement. Sometimes a slight degree of anæsthesia and analgesia can be discovered by ordinary tests, but often the disturbance of sensation is purely subjective. There is rarely any inco-ordination, and paresis is usually wanting. It may develop in the feet as well as in the hands, making walking more or less disagreeable, and adding to the discomfort of the patient. The affection is of indefinite duration; often subsiding quickly under treatment, sometimes baffling all attempts to arrest it. For a time it was considered a purely functional affection, then a spinal-cord disease;[1] but now, in the light of the parallelism between the symptoms mentioned and those which are characteristic of multiple neuritis, we cannot but consider it a slight form of this disease.

The second of the peripheral neuroses which must be referred to peripheral neuritis is intermittent paralysis. Cases of sudden paraplegia, lasting a few hours and passing off as rapidly as it appeared, have been observed too frequently to admit of any doubt. Westphal has described such a case in which no cause could be found. Gibney reported some cases presumably due to malaria. Others have thought the disease of functional character, either central or peripheral. But in the light of recent observations upon infectious cases of multiple neuritis it becomes evident that these sudden, transient paraplegiæ find their adequate explanation in such an affection.

And, lastly, there are numerous cases of indefinite nervous symptoms, pain of various kinds, formication, and odd sensations grouped under the indefinite term numbness, flashes of cold and heat accompanied by actual changes in the temperature of the part, or only by apparent vascular irregularities, slight spasms or tremors, functional weakness, with sense of fatigue not reaching the grade of paresis, and many equally obscure manifestations of disturbed function in various parts of the body, which receive their best explanation in the theory of multiple neuritis.[2]

And since it was one of the objects of the founder of this lectureship to determine the true nature of such peripheral neuroses, it is with the greater interest that we examine the disease to which they must be assigned.

In the present stage of progress in the history of neuritis much attention is being given to the etiology of the disease. Cases which, from their causation, were formerly separated are now found to be closely allied in their pathology. Thus the forms of paralysis occurring after the ingestion of various poisons, such as arsenic, lead, bisulphide of carbon, and alcohol, are known to be due to a common pathological change. The various kinds of sensory and motor disturbance occurring as complications of the acute diseases, diphtheria, variola, typhoid and typhus fevers, and severe malarial fever, are traced to a lesion in the peripheral nerves. Tuberculosis is known to predispose to neuritis, and many cases formerly supposed, without question, to be produced by central or meningeal affections of a tubercular character are now assigned to a peripheral cause. It is a question whether syphilis will cause a simple degenerative neuritis, but syphilitic affections of the nerves are easily recognized and well known. Nor can the nervous system escape the action of those micro-organisms which are now recognized as the constant cause of many diseases. There is an epidemic form of multiple neuritis, fortunately not prevalent in this country, but occasionally imported here in the form of sporadic cases, known as kakke, or beriberi, the bacillus of which has recently been discovered and cultivated. And, lastly, there is a class of cases, of supposed spontaneous origin, in which cold or over-exertion are assigned as causes, but which need further investigation in regard to their etiology.

---

[1] Ormerod, St. Bart. Hosp. Rep., 1883; Sandby, Lancet, 1884; W. Sinkler. Phil. Med. Times, 1884.

[2] Dana, Acroneuroses, N. Y. Med. Rec., July, 1885.

It is evident from this array of causes that several conditions formerly separated from one another because of the different circumstances of their occurrence are really forms of the same disease. But while they may be brought together upon a pathological basis, and while all have many symptoms in common, each of the forms of neuritis presents certain distinct features. Without dwelling too long upon any single variety, it will be necessary to review the different classes of cases in order to obtain a clinical picture of each. And such a review, together with the analysis of the symptoms of multiple neuritis, the discussion of its diagnosis from other diseases, its prognosis, and the treatment, will occupy us at the next lecture.

LECTURE II.

MR. PRESIDENT, LADIES AND GENTLEMEN OF THE PATHOLOGICAL SOCIETY :

IN the last lecture we studied the history of multiple neuritis, and incidentally obtained some clinical pictures of various forms of this disease. We also discussed its pathology and its etiology. This evening we must look more carefully at the symptoms presenting themselves in the different varieties of the affection.

An etiological classification of the cases of multiple neuritis appears to be the one most useful to the clinical observer, and as the forms vary considerably in their symptoms, we shall perhaps arrive at a more definite knowledge of the disease if we consider each of the classes separately. It is possible to distinguish—

1. Toxic cases, due to poisoning by alcohol, arsenic, lead, and bisulphide of carbon.

2. Infectious cases, due to the direct action upon the nervous system of the infectious agents, producing diphtheria, variola, typhoid and typhus fevers, severe malarial fever, and tuberculosis; to which must be added the agent causing the epidemic form of neuritis known as kakke or beriberi.

3. Spontaneous cases, due to uncertain causes, among which cold, and exposure to damp and wet, and to overexertion, may find a place.

I. TOXIC CASES.—(*a*) *Multiple neuritis, due to poisoning by alcohol.* While alcoholic paralysis has been universally recognized for many years, it is only within a short time that the symptoms and pathology have been brought into a logical connection. It is to Moeli,[1] of Berlin, and Dreschfeld,[2] of Manchester, England, that we owe the most important recent contributions to the knowledge of this form of neuritis, though the work of Henry Hun,[3] of Albany, and of Bernhardt,[4] of Berlin, cannot be passed over without recognition.

It will be remembered that among the first cases of multiple neuritis in which an autopsy was made, viz., those of Lancereaux,[5] the disease was traced to alcoholism. Lancereaux deserves the credit of demonstrating the pathological basis of alcoholic paralysis, for the descriptions of its symptoms previously given by Jackson and Huss were not accompanied by post-mortem records. But Lancereaux's observations were for many years overlooked by physicians outside of France; and in England, where the disease is well known, it was ascribed, as lately as in 1883, to a spinal lesion.[1] But in 1884 Dreschfeld combated this theory and called attention to its true pathology, showing that cases formerly supposed to be spinal paralysis were really due to an affection of the peripheral nerves.

---

[1] Moeli: Charité Annalen, 1884.

[2] Brain, Nos. 26 and 32.

[3] Amer. Jour. Med. Sci., April, 1885.

[4] Zeitsch. f. Klin. Med., 1886

[5] Gaz. Hebdom., 1881.

All observers have emphasized the fact, already noted by Jackson, that the disease is especially frequent among females. Males are not, of course, exempt from alcoholic paralysis, but in them the poison seems more liable to manifest itself by acute cerebral symptoms than by those of disease of the peripheral nerves. It is especially frequent among those persons in the higher classes whose nervous organism is highly developed, and who lead a comparatively inactive life. It seems not at all improbable that sedentary habits predispose an alcoholic drinker to this disease, and hence active workers, male or female, though taking an equally large amount of liquor as the luxurious drinker, escape. All alcoholic drinks are not equally prone to produce paralysis. It is the spirituous liquors—brandy, whiskey, gin, and rum, and the liqueurs, absinthe, vermouth, etc.—which are dangerous. And it is only after these drinks have been consumed in large amounts and for a considerable length of time that neuritis develops. Its onset, though often apparently very sudden, is usually gradual. For months the patient has suffered from chronic gastritis, insomnia, general neuralgic pains, or severe pains in the joints or limbs, and from tremor and a certain feebleness in movement, when all at once her legs give way beneath her, and after the sudden fall she finds herself unable to rise. Thus a patient of my own, after a year of such premonitory symptoms, was seized with paralysis quite unexpectedly when getting out of bed in the night. This paralysis soon becomes complete in the feet and legs below the knees, and may advance up the thigh. It next attacks the hands and forearms, and while in all extremities it is often greater in the extensors than in the flexors, in some cases both groups of muscles become entirely helpless. This has been the case in three patients under my care. The paralyzed muscles are flabby and soon become atrophied, they have no excitability to mechanical irritation, and the tendon reflexes are lost. They fail to react to a faradic current in the majority of cases, though occasionally a very strong current may produce a response. When galvanism is applied the reaction of degeneration is found to be present. No stimulus can be given to the muscle by sending a current through its nerve, and the positive pole produces more marked contractions with an equal current than the negative pole when placed on the muscle, and then it is only a slow or vermiform movement, not the quick jerk of health. It is also found that strong galvanic currents have to be used to produce any contraction at all. The paralysis of the muscles may advance rapidly in severe cases, involving the motor cranial nerves, the muscles of the trunk, and lastly the diaphragm, thus causing death. More frequently, however, it is arrested when only the distal parts of the extremities are involved, and then it gradually subsides until recovery is complete. The position assumed by the paralyzed limbs has been thought to be almost characteristic. There is dropped wrist, quite similar to that seen in lead-palsy, and also dropped foot, due to the falling forward of the foot from its own weight, since the anterior tibial muscles are weak. This deformity is increased by the fact that the patients lie in a recumbent posture with the feet extended; and when the flexors of the toes are but slightly affected, as sometimes is the case, their unopposed contraction serves to exaggerate the malposition. If there is entire paraplegia the legs and thighs may both be extremely flexed, so that the heels touch the buttocks, but this is exceptional. While the dropped wrist is the usual deformity of the hands, cases are recorded in which the paralysis was limited to single muscles and to muscles supplied by single nerves. Thus Lilienfeld describes a weakness of the extensors of the thumb, fourth and fifth fingers, and Leudet mentions a paralysis of the ulnar nerve. In one case of my own the *main en griffe* was the deformity noticed, indicating a weakness of the interossei only. Such cases as those of Hun,[1] where a facial paralysis was seen, or of Lilienfeld,[2] where double abduceus paralysis occurred, are certainly rare, and still more so are those in which irregular, rapid pulse has been ascribed to an affection of the pneumogastric.

---

[1] Wilks: Dis. of Nerv. System, p. 272.

To the physician these motor symptoms, and the œdema, occasional lividity, profuse sweating, and glossy skin, so often associated with them, are very noticeable. But the patient suffers far more from the disturbances of sensation. In the description of Jackson the pains were graphically portrayed. They are the cause of terrible agony, sufficient to produce insomnia, and wearing seriously upon the endurance of the sufferer.

---

[1] Hun: Loc. cit.

[2] Lilienfeld: Berl. Gesellsch. f. Psych. und Nervenkrankheiten, July 15, 1885 see also Richard Schultz, Neurol. Centralbl., Nos. 19, 20, 21, 1885.

In addition to pain, hyperæsthesia is not infrequently observed. It is usually quite extensive in the legs, though in cases of poisoning by absinthe it has been limited to the soles of the feet. The muscles, as well as the skin, are sensitive to handling and to pressure, and marked tenderness in the course of the nerves is always elicited by examination. In one of my cases, soon after the onset, the patient could not bear to be touched or moved, though perfectly unable to help herself. Charcot goes so far as to say that muscular sensitiveness associated with flaccid paralysis is pathognomonic of alcoholism.

Paræsthesiæ are always complained of. Numbness, tingling, and formication are frequent. In one of my patients the sensation was as if heavy bracelets were around the wrists, and as if very tight drawers were on the legs. At other times she felt as if the limbs were swollen and as if the skin were about to burst. Such sensations may cease as the case increases in severity, and give place to a total lack of sensation in the parts. They return, however, with advancing recovery, and are among the last symptoms to disappear.

Abolition of tactile sense, and to some degree of muscular sense, is the rule after the paralysis is developed. Temperature-sense and the perception of pain are never wholly lost, but may be delayed in transmission. The anæsthesia may be limited to irregular areas, and may be only in the cutaneous distribution of one nerve, but is usually found over the entire distal part of the paralyzed limb. Usually the cutaneous reflexes are preserved. The loss of muscular sense is, in some cases, so marked a symptom, and one of such early occurrence, that Dreschfeld distinguishes a class of cases which he terms ataxic rather than paralytic. And this distinction is perfectly justifiable, for in many cases it is the inco-ordination which attracts the attention of both the patient and the physician. It is this class of alcoholic cases which may be mistaken for locomotor ataxia, and which have been named by French writers pseudo-tabes alcoholique.[1] But ataxia is not exclusively limited to this class of cases. It may be present in some degree in cases of paralysis, and during recovery from paralysis the deficiency in co-ordinating power may become evident, and appear to retard the progress of the case. Nor are the cases of ataxia, on the other hand, free from paralysis.

And this fact is proven by the observations of Westphal[2] and Charcot. For, by contrasting the walk of a true ataxic patient with that of an ataxic alcoholic patient, they have each discovered several points of difference. The ataxic patient throws the foot forward with undue violence, the toe lifted high in air, and brings first the heel down forcibly and then the entire foot. The alcoholic, however, has some weakness in the muscles of extension and cannot raise the toe. He therefore lifts the foot high in order to step over the hanging toe and not to trip on it, but the motion is made without undue force. He then throws the foot forward, in order to throw the toes up and get them out of the way as he brings the foot down to the floor. The motion is awkward and has an appearance of one stepping over high obstacles, but it is a voluntary attempt to remedy a deficient power—not the involuntary awkwardness of a man unable to manage strong muscles. There may be in both patients some tottering, and swaying when standing with the eyes closed, the so-called Romberg symptom is common to both locomotor ataxia and alcoholic neuritis. Dejerine describes the following case, in which the symptoms resembled those of tabes :

---

[1] Dejerine: Arch. de Phys., 1884.

[2] Westphal: Ueber eine bei chronischen Alcoholisten beobachtete Form von Gehstörungen, Charité Annalen, 1879. Charcot: Leçons, Progrès Médicale, 1886.

Observation VIII.—Male, forty-two, a hard drinker, complained for four months of shooting pains in the legs, and noticed a gradually increasing difficulty in walking, which was worse in the dark. He had also found his feet swollen at times, and had some difficulty in micturition. Examination on admission to the hospital showed a high grade of ataxia in walking, Romberg's symptom, loss of tendon reflexes, anæsthesia almost total of entire body, considerable analgesia with retardation of sensations of pain, temperature-sense being preserved, and loss of muscular sense in the legs. The muscles were slightly feeble and a little diminished in size. It could hardly be said that there was paralysis or atrophy. The diagnosis made was locomotor ataxia of an unusual type. The onset was so rapid, the anæsthesia so extensive and severe, the paresis of the limbs, though slight, so evident, that it differed from a typical tabes. But no other diagnosis could be made from the symptoms. The patient died of chronic nephritis six weeks after admission. The autopsy showed the spinal cord to be normal. But in the cutaneous, and to a less degree in the intra-muscular branches of the nerves, as well as in the distal parts of the nerve-trunks, a high grade of parenchymatous neuritis was found. The nerve-tubes had lost their double contour, the myelin sheath was segmented, the protoplasm was increased, the nuclei multiplied. The nerve-trunks on the plexuses and the nerve-roots were normal. The nerves of the legs were more deeply involved than those of the arms.[1]

---

[1] Dejerine : Arch. de Phys. Normale, 1884. For other ataxic cases, see Dreschfeld, loc. cit

The second case, in a female forty-nine years of age, presented almost identical symptoms and lesions.

The special senses are occasionally affected in cases of alcoholic paralysis. Amblyopia has been observed, and also defective vision from central scotoma. The field of color-vision is often contracted, even when sight is preserved. There may develop a true optic neuritis, evident to the ophthalmoscope, and this may go on to optic-nerve atrophy.[1] Inequality of the pupils is frequently seen, as is also a moderate contraction of the pupil. All these eye symptoms, occurring as they may in a case of the ataxic variety, make a differential diagnosis from locomotor ataxia difficult. The Argyle Robertson pupil (which contracts in accommodation but not to light) has not been seen in alcoholic cases, while it is an early symptom of tabes.

One feature of alcoholic paralysis remains to be noticed, viz., the cerebral symptoms. These are hardly ever wanting. There is at first the excitement rising to the degree of active delirium, with illusions and hallucinations of the various senses; there is the insomnia which so soon exhausts the patient if it is not remedied; there is the loss of memory, especially of recent occurrences; and the lack of power of attention or concentration which prevents intelligent conversation. The indifference to bodily wants may be so great as to lead to uncleanliness, and since paralysis of the sphincter is the rare exception, incontinence is usually to be ascribed to the mental state. It is useless to attempt to get any reliable history of their illness from these patients. Their statements are unintelligible or unreliable. And here it may be well to notice a symptom first remarked by Strümpell.[2] These patients will relate occurrences as having happened recently with much elaboration of detail, when as a fact the story is entirely a product of their imagination. Thus one patient of my own, who had been confined to bed for many days, told me one afternoon that she had been out to see an eminent gynecologist during the morning; had gone to his office and waited for him several hours; had seen other patients there, and finally had been told by the doctor's brother that he would not return in time to see her, so she had come home again. And this was all related in apparent good faith, so that I have no doubt that she believed that what she said had occurred. With the possibility of such delusions in view, it is evident that the statements of these patients cannot be accepted regarding anything, especially as to their own history.

One patient, who was admitted to Bellevue Hospital during my service there, told me a different history of her case every day for a week, and it was only by interviewing her friends that the correct account was obtained.

---

[1] Brissaud: Des paralysies toxiques, p. 31. Paris, 1886.

[2] Strümpell: Arch. f. Psych., 1883, xiv., 339.

The course of alcoholic neuritis is quite uniform. After a sudden onset the symptoms rapidly advance to a high degree, which is reached in a week or two from the beginning of the paralysis or ataxia. Then they may increase further, and cause death by respiratory paralysis. Usually they remain stationary for a time, and then gradually subside, the entire duration being from two months to a year. Individual muscles regain their power, tone, firmness, and electrical reaction slowly, and during recovery the tingling and numbness in hands and feet may be severe. In a few cases the muscles become contractured, and permanent deformities, only to be overcome by long-continued massage or by operative measures, develop. When the fact is considered that those who recover rapidly rarely fail to resort again at once to the use of stimulants, and thus expose themselves to the danger of a relapse, the ultimate fate of the chronic cases is hardly more serious than that of those who get well.

As examples of the paralytic form the following cases are cited:

Observation IX.—A baker, aged forty-seven, phthisical, a moderate drinker, had suffered for several years from numbness of the hands and legs; and for a year before the acute invasion of his illness from a slight weakness of the legs and uncertainty of motion in walking. This weakness became much worse during the spring of 1881, so that all summer he was confined to his room, though he managed to walk a little. In November, after an uneasy night, during which he was delirious, it was found that he could not move his legs at all, and that his arms were powerless. He was brought to the hospital November 25th, when a total paralysis, with atrophy and loss of tendon reflexes, was discovered in legs and arms, as well as slight disturbances of sensibility in the legs, consisting of a delayed sensation of pain. His mental condition was a weak one, but he answered questions rationally. The face was in no way affected. No involuntary evacuations. Electric examination showed a great diminution, and in many muscles total loss, of faradic excitability when the current was applied both to nerve and to muscle. With the galvanic current few contractions could be elicited by exciting the nerves, and by exciting the muscles the slow contractions characteristic of RD,[1] as well as the predominance of AnCC over

---

[1] RD = reaction of degeneration; AnCC = anode closure contraction; KCC = cathode closure contraction.

KCC were present. The mechanical excitability of the muscles was not impaired. There were no pains. During the following three months there was little change in the condition, except that his mind was not clear, and that he told extraordinary stories of having been out walking, of having made purchases, etc., when, in fact, he could not move from the bed.

His pulse became more rapid during this time, averaging 120 per minute. In February, 1882, the legs had become œdematous, and there was marked analgesia in them, except on the soles of the feet, which were hyperalgesic; sensations of touch were, however, readily perceived. Though he had a sanguineous dysentery, there was no incontinence. On the 12th there developed suddenly great difficulty in respiration, due to suspension of all diaphragmatic breathing, and the next day he died. An ophthalmoscopic examination on the last day showed an atrophy of both optic nerves on the outer side of the eyes.

Autopsy: Brain and cord normal. No macroscopic changes in the nerves. A microscopic examination, however, showed the presence of a very high degree of degeneration and degenerative atrophy in the nerve-fibres. The myelin had divided in large and small drops, containing fat-granules; the degenerated mass within the Schwann sheath, consisting of myelin and axis-cylinder, was seen in various stages of absorption, and in spots the empty Schwann sheath alone remained. No inflammatory exudation was present, and the endoneurium and perineurium appeared to be normal. The muscles showed evidence of degenerative atrophy. The anterior nerve-roots were normal.[1]

The lesion here resembled exactly that in Joffroy's case, already cited. It was similar in the following case:

Observation X.—Female, who had recently indulged in alcoholic excesses and had suffered from gastritis and delirium tremens; complained for some days of pain in her legs and numbness in hands and forearms. When seen by Dr. Webber the pains in the limbs were severe, and she could not turn in bed without help, her knees were drawn up, and attempts to straighten them caused pain. There was loss of power in extensors of the wrist and hands, and the fingers could not be shut perfectly. There was general tenderness over the muscles of the extremities, and pressure along the nerves was very painful. Her throat was sore, but she did not have diphtheria. She spoke in a whisper and with difficulty. Three days later anæsthesia of the feet and hands appeared, and the tendon reflexes were lost. The pain became so severe that morphine was given to control it. The paralysis became more complete, the anæsthesia increased, incontinence of urine and fæces developed, she appeared to be very feeble, and respiration became wholly thoracic. Seventeen days after admission to the hospital she died.

---

[1] Strümpell: Arch. f. Psych., 1883, xiv., 2.

Autopsy showed brain and cord normal. The chief nerve-trunks of the extremities, and the phrenic and pneumogastric, were in a condition of degeneration, the distal ends being affected to a greater degree than the central portions. "The simplest change found was in the medullary sheath at Ranvier's constrictions, without break in the axis-cylinder, and without increase of nuclei." In some nerves extensive degeneration was found, the medullary sheath being broken up into fatty fragments, the axis-cylinder discontinuous or destroyed, and the number of nuclei increased. In the fibres which were most altered was found an increase of nuclei; and apparently small masses of protoplasm, not nuclei, were tinted in the midst of the granular débris. Nearer the root of the nerves axis-cylinders were more numerous. In the sciatic the sections from the highest part of the nerve, which had been preserved, were still diseased in a small proportion of their fibres.[1]

A very similar case, with the same changes in the nerves, is recorded by Müller.[2]

The following case, under my own care, terminated favorably:

Observation XI.—Male, aged thirty-six, a hard drinker, after an attack of gastritis became paralyzed suddenly in both legs. A few days after this, both hands became useless, so that he was completely helpless. His wife says that his memory is quite gone. On admission to Bellevue Hospital, September, 1880, seven weeks after the onset, he complained of pain in all four extremities, and of tenderness in the muscles and joints when these were handled. The upper extremities were almost totally paralyzed below the elbows, the only motion possible being a slight flexion of the fingers. There was wrist-drop on both sides. The lower extremities were totally paralyzed below the knee, and the feet hung down motionless. The muscles of the thighs were also affected, for although he could pull his legs up in bed, it was with great difficulty that they could be straightened out. All the paralyzed muscles were atrophied, and the faradic reaction was lost in the extensors and greatly diminished in the flexors. The galvanic reaction was not tested by me from lack of apparatus. Tactile sense was considerably impaired, but the senses of pain and temperature and muscular sense seemed to be natural. The knee-jerks were lost. When an attempt was made to have him stand, the feet were pushed out and the knees doubled under him at once. Fibrillary twitchings were very noticeable in the atrophied muscles, and a marked tremor of the tongue was seen. The facial and ocular muscles were not affected, and the special senses were normal. There was no incontinence of urine and no symptom of thoracic or visceral disease. His mind was much impaired. He talked in a rambling manner, laughed much, could not fix his attention, and his memory was so poor that no reliance could be put in his statements. Under treatment by iodide of potash, and complete cutting off of all stimulants, he began at once to improve. In two months from the time of admission he was able to walk, and one month later he was discharged perfectly well.

---

[1] S. G. Webber: Arch. of Med., 1884, xii., 33-49.
[2] F. C. Müller: Arch. f. Psych., 1883, xiv., 3.

These cases are sufficient to present a clinical picture of the symptoms and course of the forms of alcoholic multiple neuritis. The differential diagnosis, elements of prognosis, and means of treatment will be considered when other classes of the disease have been studied.

(*b*) *Multiple neuritis due to poisoning by arsenic.*[1]—It has long been known that an occasional result of arsenical poisoning is the development of paralysis, but it is only within the past four years that the fact has been determined that the symptoms in these cases are due to an affection of the peripheral nerves. Had the fact of the peripheral origin of alcoholic paralysis not been already proven, it is probable that the theory so long in vogue, that arsenical nervous symptoms were due to spinal lesions, would still prevail. But there is such a similarity between the two sets of cases, that it is impossible to ascribe them to other than the same pathological condition. Autopsies in support of the position that the peripheral nerves are involved are few in number, but in several cases the lesion has been found. The observations on record of spinal lesions are, it is true, more numerous; but when these are compared it is found that different lesions have been discovered in different cases, so that there is no single pathological change in the spinal cord which is constantly produced by arsenic. Further, some of the changes described in experimental cases in animals (viz., vacuolization of cells) are due to imperfect hardening of the specimens. It must, therefore, be admitted that multiple neuritis may be due to arsenical poisoning.

---

[1] Scolozouboff: Arch. de Phys., 1884, p. 323; Imbert Gourbeyer: Des Suites de l'empoisonnement arsenical, Paris, 1881; Seeligmüller: Deut. Med. Woch., 1881, p. 185: Lancereaux: Gaz. Hebdom., 1881, p. 719; Jaeschke: Thèse Breslau, 1882, Ueber Lähmung nach acuterar senik Vergiftung: Levin: Schmidt's Jahrbuch, Bd. 165, p. 239; Da Costa: Phil. Med. Times, 1881, p. 385; C. L. Dana: Brain, January, 1887; Brissaud: Paralysies toxiques, Paris, 1886.

The changes produced in the nerves are so exactly similar to those already described, that there is no need of a recital of the pathological process.

The nervous symptoms produced by arsenic have been thought to vary somewhat, according as the ingestion of the poison has been a sudden or a gradual one. Brissaud claims that if there is slow poisoning, as, for example, by the long-continued use of Fowler's solution, paralysis is rather the exception, and is not severe, it is diffuse and transient; while other symptoms, such as gastro-enteritis, trembling, delirium, and aphasia attract the chief notice. If there is acute poisoning from an overdose of arsenic, he holds, on the other hand, that paralysis ensues either during the period of active symptoms of poisoning, or soon after. The observations of other equally careful authors do not entirely support this view of Brissaud; for in two cases of Dana, one of acute, the other of chronic poisoning, very similar symptoms of paralysis and ataxia developed.

The description which has been given of alcoholic paralysis might almost be repeated for arsenical paralysis. There is the same limitation of the affection to the muscles of the distal parts of the extremities, the extensors being chiefly affected, and the weak muscles are flaccid, soft, and atrophied. There is a partial reaction of degeneration. The tendon reflexes are abolished; skin reflexes are preserved. There is often a marked tremor. The paralysis may begin either in the feet or in the hands, is usually bilateral, but has been in four cases of the hemiplegic type. The same dropped wrist and dropped foot are seen as in alcoholic cases. Disturbances of sensibility are prominent symptoms; burning, tearing, shooting pains; formication, tingling, muscular, and arthritic pains and tenderness are associated with hyperæsthesia, and this may be followed by irregular patches of anæsthesia. The muscular sense is usually impaired, and so much so in some cases that an attempt has been made to establish a distinct class of cases as arsenical ataxia, or pseudo-tabes arsenicale. In these cases the inco-ordination of hands and feet, Romberg's symptom, and an awkward gait are very noticeable, so that tabes may be suspected until the history makes the causation evident. In a case of Dana's the patient could not tell the position of his limbs or distinguish between weights differing one to forty. In Scolozouboff's case the walk resembled that described by Westphal in alcoholic tabes, and was easily distinguished from a real ataxic gait. Seeligmüller and Dana, however, affirm that the gait in their cases was like that of locomotor ataxia. Occasionally œdema of the extremities, cyanosis, unusual sweating, and extensive desquamation indicate an implication of vaso-motor and trophic nerve-fibres. In the most severe cases contractures in a flexed position developed in the paralyzed limbs. The sphincters are never involved. The duration of arsenical paralysis is somewhat greater than that of ataxia. Either condition may last several months, but occasionally the recovery is complete in a few weeks. Frequently, after the power has returned, the patient suffers for months from numbness and tingling in the extremities, which is sufficient, as in a case under my own observation, to interfere with the finer motions, to disturb the sleep and cause constant discomfort. According to Gerhardt's statement (cited by Dana), ninety-seven per cent. recover wholly.

(*c*) *Multiple neuritis due to poisoning by lead.*—It is not my purpose to enter upon any description of the various forms of lead palsy, which are familiar to every practitioner. Nor is this the proper place for a discussion regarding the various theories of the pathology of the disease. It is only necessary to call attention to the fact that there are now on record a number of autopsies in cases of lead paralysis in which the lesion has been found in the peripheral nerves. In a recently published case of Schultze[1] there was found a very marked atrophy and disappearance of nerve-fibres in the trunk of the musculo-spiral nerve, below the point where the branch to the supinator longus was given off. This decreased in intensity centrally, so that at the brachial plexus no anomaly was found. It increased in intensity toward the termination of the nerve in the muscles. The spinal cord was normal. This is simply a type of a number of recently published cases. On the other hand, there are numerous cases of this disease in which decided spinal lesions have been found—so numerous that many writers always ascribe the disease to destruction of certain groups of cells in the anterior cornua of the spinal cord. It must be admitted, therefore, that in lead we have a poison which, under certain circumstances, affects the spinal cord, and under other circumstances produces neuritis. It may be claimed that the same is true of alcohol and arsenic, and in showing that these poisons can cause neuritis there is no intention of defending the position that they never affect the central nervous system. But it is important to notice that the central organs are rarely involved. The limitation of the neuritis in lead palsy to the motor nerves makes the clinical picture in some cases resemble closely that of spinal disease. In illustration of this the following case is cited:

---

[1] F. Schultze: Ueber Bleilähmung, Arch. f. Psych., xvi., p. 791.

Observation XII.—A male, aged thirty-six, by occupation a dyer, had suffered from numerous attacks of lead colic, followed by general muscular weakness, which on one or two occasions was so great as to be a general paralysis rather than a simple weakness, and had finally developed a paralysis of the extensors of arms and legs, when admitted to the hospital, where he spent the last three years of his life. During this time his paralysis varied somewhat in degree, but was never entirely recovered from. The muscles affected were the extensors of arms and legs. These were much atrophied, and showed a loss of faradic reaction, and a characteristic change in the galvanic reaction, the anode producing stronger contractions than the cathode, and all contractions being slow. There was also a considerable degree of œdema in the extremities, especially in the legs. Spasms in the unparalyzed flexors occurred on one occasion for one day. The patient suffered also from the symptoms of cardiac disease and phthisis, to which he finally succumbed.

The autopsy showed no changes in the brain or spinal cord. The muscular branches of the peripheral nerves, although presenting no macroscopic changes, were found in a state of extreme degeneration when examined microscopically. Side by side with normal fibres were to be seen fibres, unstained by osmic acid, wholly destitute of myelin sheath. There was no fatty degeneration. Between the fibres a thick, finely fibrillary connective tissue, with small cells, was found. Transverse sections of the nerve-trunks showed a large number of small fibres scattered among the normal fibres, but not collected into bundles, and it was often difficult to distinguish naked axis-cylinders from connective-tissue fibres. The lesion was an increase in the connective tissue of the endoneurium with simple atrophy of the nerve-fibres of different grades.[1]

But in a certain proportion of the cases of lead palsy there are marked sensory disturbances, consisting of severe pains, and anæsthesia, with numbness. In these it is possible, especially if tenderness along the nerves and in the muscles is present, to make the diagnosis of multiple neuritis. The history of the following case, under my care, coincides so closely with that of other toxic cases of neuritis that the diagnosis could not be mistaken.

---

[1] Eisenlohr: Deut. Arch. f. klin. Med., 1880, xxvi., p. 543.

Observation XIII.—Male, aged thirty-eight, after suffering from an attack of lead colic, began to feel numbness and pain in his legs and feet, which soon extended to his hands and forearms. This steadily increased for two weeks, and to it was added paralysis of the extensors of both hands and both feet, so that at the end of that time he was unable to use his hands or to stand. With the paralysis there was a rapid atrophy of the muscles, and a decline in the faradic excitability. The muscles became more and more tender, and the spontaneous pains gradually increased until it was necessary to use opium freely to quiet them. By the end of a month a well-marked anæsthesia had developed below the knees, and it was noticed that the atrophied muscles were in a constant tremor. There was wrist-drop and foot-drop on both sides, and all reaction to faradism ceased. It required a very strong galvanic current to produce contraction, and AnCC was greater that KCC. The knee-jerk was preserved. The muscular sense was impaired. For five months his condition remained stationary, in spite of treatment by strychnia, iodide of potash, massage, and electricity. Then a gradual improvement set in, the pains became less severe, sensation returned, the muscles regained their contour and strength, and finally the electric contractility returned to the normal standard. About a year from the onset of the symptoms, the recovery was complete.

(*d*) *Multiple neuritis from poisoning by sulphide of carbon*[1] *and from illuminating gas* has been suspected, and the similarity of the symptoms in such cases to those already described is quite remarkable. But as there are no autopsies to substantiate the theory, this cause is merely mentioned.

II. MULTIPLE NEURITIS CONSEQUENT UPON INFECTIOUS DISEASES.—There are a number of infectious diseases which are especially liable to be followed by the development of nervous symptoms. These are diphtheria, variola, typhoid, typhus, and scarlet fever, malarial fever, and tuberculosis. The nervous affection usually appears shortly after the period of convalescence in the acute fever. The disease may consist of a simple paralysis of the muscles in the region of distribution of a single nerve. It may affect several nerves on both sides of the body symmetrically. It may even paralyze two or more limbs. It occasionally produces sensory as well as motor symptoms in a single nerve-trunk. It may even cause a general sensory and motor paralysis of as widespread and complex a kind as that produced by chronic alcoholism.

---

[1] Ross: Medical Chronicle, January, 1887.

Sometimes the symptoms are chiefly of a sensory kind, and consist exclusively of pains, numbness, and anæsthesia, or hyperæsthesia, in the legs, or of a loss of the muscular sense, in which case a true ataxia is the most noticeable symptom. Thus it is evident that the poison of an infectious disease may act as powerfully upon the nervous system as any other form of poison known.

It is only since the clinical pictures presented by multiple neuritis have been recognized that a question has arisen as to the part of the nervous system affected in these cases. Formerly all such phenomena were referred to central lesions; and undoubtedly in many cases this was justified, since autopsies are not wanting to prove that anterior poliomyelitis, diffuse myelitis, and hemorrhages into the cord and brain may follow the acute fevers. There are, however, many conditions which do not correspond to the types of disease produced by central lesions and which recover with a rapidity impossible were the brain or spinal cord involved. It was these cases which raised the question of some possible affection of the peripheral nerves. And careful investigation has been rewarded by the actual discovery of lesions in them.[1]

(*a*) Diphtheritic paralysis is probably more common than any other of these forms of neuritis. As is well known, it is usually the soft palate to which the paralysis is limited, and as a result difficulty in swallowing and in speech are the most prominent symptoms. Bernhardt has found[2] that in the large majority of cases of this kind there is a loss of the patella tendon reflex, but whether this indicates any general affection of the peripheral nerves he does not venture to state. The limitation of the paralysis to the palate has been explained by supposing that the poison of the disease has a direct action upon the terminal filaments of the nerves, which in this position are, as it were, dipped constantly in the poison. This theory is supported by a case in which paralysis of the abdominal muscles was associated with diphtheritic inflammation of the navel in a new-born child. But the more serious cases prove that through the blood the poison may be carried to nerves far removed from the seat of the diphtheritic inflammation.

And the following cases demonstrate the wide-spread symptoms which such general poisoning can produce, as well as the nature of the lesion.

Observation XIV.—Marie S——, aged thirteen, after suffering for a few days from a slight sore throat, found herself unable to see clearly because of the development of a paresis of accommodation. There followed a slowly progressing ataxia of the upper, and later of the lower, limbs, with uncertain gait, disturbance of touch, pain, temperature, and muscular senses, with delayed sensation of pain and loss of tendon reflexes. At first she felt shooting pains in the limbs, but later these ceased. There was at no time tenderness of the nerves to pressure. After three months bulbar symptoms appeared, atrophy, with paresis of the tongue, difficulty in swallowing, spasms of coughing, weakness of voice, and paralysis of the palate. The muscles of the hands began to atrophy about the time that the bulbar symptoms commenced, and soon were useless. In the atrophic muscles the faradic reaction was absent, the galvanic much reduced, and very slow. Nine months after the onset she died of pneumonia.

---

[1] Pitres et Vaillard: Rev. de Médecine, 1885, p. 986; 1886, p. 193 et p. 574, Des Névrites Périphériques chez les typhoides, les tuberculeux et les tabétiques; also, Arch. de Neurol., 1886, p. 337, De la névrite segmentaire.

[2] Virch. Archiv, 1885, Bd. 99, s. 393.

The autopsy showed the brain and spinal cord to be normal. In all the peripheral nerves of the extremities, as well as in the hypoglossal and recurrent laryngeal nerves, well-marked atrophic degeneration was found, with destruction of axis-cylinders and medullary sheaths, and with thickening and increase of nuclei in the connective-tissue sheaths.[1]

Observation XV.—Male, aged seventeen, after an attack of diphtheria grew gradually weaker and weaker, developed paralysis of the uvula and paralysis, with some atrophy, in the muscles of all the limbs, and a slight paralysis of the face and tongue. There was no real ataxia, the muscular sense being normal, but the weakness of the muscles interfered with all accurate movement and caused trembling. The eyes were not affected. There was a slight decrease of tactile sense all over, but pain-sense was slow and imperfect. The reflexes were lost, electric reaction was normal in all muscles except the palate, in which RD was found. The sphincters were unaffected. The paralysis involved the respiratory muscles three weeks after the onset of the disease, and caused his death.

The autopsy showed extensive parenchymatous neuritis in all the peripheral nerves. The various stages of degeneration were found side by side in many nerves, and all degrees of degeneration were discovered. The nerves of the extremities, as well as the phrenics, showed these changes. Some nerve-fibres had simply lost their myelin sheath, others were reduced to a series of granular corpuscles; others consisted only of the empty Schwann sheath, whose nuclei were numerous. In many nerves

---

[1] Kast.: Deut. Arch. f. klin. Med., 1886 Bd. xl., s. 41.

the lesion only involved a few segments, and was not continuous through the entire length. At places circumscribed thickening of the endoneurium produced nodular appearances in the course of the degenerated nerve. Here congestion and emigration of leucocytes were found. The perineurium was unaffected. In almost all the muscles of the extremities and trunk such degenerated nerve-fibres were found. The cutaneous nerves were similarly affected, but to a less degree. The nerve-trunks were but slightly affected, as were also the nerve-roots. The spinal ganglia and cord were normal.[1] The limitation of the degeneration to single segments has also been noticed by Gombault and by Pitres et Vaillard.[2]

Observation XVI.—A boy, aged eight, developed paralysis of the soft palate one week after an attack of diphtheria. This continued for one month, when there suddenly appeared a weakness of all the extremities, and some disturbance of sight. Examination of the eyes showed a complete paralysis of all the recti muscles, with double ptosis, the accommodation and pupil reactions being preserved. The right side of the face was paretic. There was a very marked ataxia in all the limbs. It was with difficulty that he could carry food to his mouth, and he walked with much staggering, and with legs wide apart. Power and nutrition of the muscles was normal, and there was no change in their electric contractility. The tendon reflexes were absent, but the skin reflexes were preserved; and there was no disturbance of sensation or of bladder and rectal functions. The symptoms all increased for ten days, and then respiratory paralysis caused his death.

It was possible to examine the brain only. The small vessels and capillaries were engorged, and the perivascular spaces full of emigrated corpuscles, while here and there small capillary hemorrhages were found. The vessel-walls were not affected, and there were no thrombi discovered. The nuclei of the cranial nerves showed no changes. Both oculo-motor nerves were the seat of very great changes. The myelin sheath of individual nerve-fibres absorbed the carmine stain. In many fibres no axis-cylinder was to be seen. In others the cylinders were swollen or atrophied, presenting variations in size. The number of nuclei of the endoneurium was greatly increased, and among them large granular corpuscles were discovered.[3] The lesion, therefore, which had caused the ocular paralysis was a diffuse neuritis.

A similar affection of the ocular muscles, associated with general weakness, has been reported by Utthoff,[1] which developed two weeks after an attack of diphtheria in a boy, ten years of age, and who at the end of three months had completely recovered. In this case all reaction of the pupils in accommodation was lost, but the light reflex remained. The eye symptoms were attended by some paresis, especially in the legs below the knees—but the muscles were not atrophied, nor was their electric contractility altered. There was a very marked tenderness along the course of all the nerve-trunks. After recovery was complete the knee-jerk returned.

---

[1] P. Mayer: Virch. Arch., 1881, Bd., 85, s. 181-226.
[2] Arch. de Neurol., xi.
[3] Mendel: Neurol. Centralbl., 1885, p. 128.

I have seen a case which resembled this quite closely, and inasmuch as paralysis of all the ocular muscles appears to be a rather rare affection, the case may be worthy of mention here.

Observation XVII.—A boy, aged thirteen, after an attack of diphtheria developed paralysis of the soft palate. One week after the paralysis of the palate he noticed a disturbance of vision, which proved on examination to be due to a total paralysis of all the muscles of the eyeballs. The pupil reflex to light was preserved; but as he could not converge the eyes there was no reaction in accommodation. This paralysis of the eyeballs came on within twenty-four hours, and at the same time he developed a very marked degree of ataxia. He swayed in standing with eyes closed, and his gait was exactly like that of a well-marked case of tabes. The motion of the hands was also disturbed, and all the tests demonstrated an extreme degree of inco-ordination. The knee-jerks were lost; the skin reflexes were present. In addition to the ataxia there was actual weakness of the muscles, which was more evident in the arms and hands than in the lower extremities. He had formication in the legs, but not in the hands, and there were no lightning pains. He had great difficulty in swallowing solids as well as liquids, which seemed to point to some involvement of the muscles of deglutition as well as of the soft palate. This condition remained stationary for about three weeks, and then all the symptoms began to pass off gradually. His recovery was not complete until six months after the onset.

The prognosis in cases of diphtheritic paralysis is usually very good, the fatal cases here cited being rarities. The treatment consists in general tonic medicines, and the application of electricity to the limbs in the same manner as in other cases of neuritis.

(*b*) Neuritis following variola is a rare complication, and the following case is the only one on record in which an autopsy proved the seat of the lesion.

---

[1] Neurol. Centralbl., 1885, p. 125.

Observation XVIII.—A young man had varioloid in November, 1881, and while convalescing, six weeks later, began to suffer from severe pains in his four extremities, especially in the joints of his arms, which were diagnosticated as rheumatic, although there was no fever. Soon after there followed a true paresis, with progressive atrophy of the muscles of forearms and legs. The muscles at the same time became very tender to touch or pressure. The tendon reflexes were much diminished. Reaction of degeneration developed in all the paretic muscles. The pains in the joints and limbs continued, but were less severe than at the outset. The sensibility of the skin was about normal. The nerve-trunks were tender to pressure. Profuse, offensive perspiration in all four extremities was a distressing symptom. There was no tendency to bed-sores, but an extensive pemphigus developed in the legs, and then the pains became more severe. The patient died in July, 1882, of pneumonia.

Autopsy showed the brain œdematous, and the cord in a state of hypostatic congestion. The pathological changes of importance were found in the nerves and muscles. The majority of the nerves of all the extremities were found in a state of degeneration and atrophy. There was marked degenerative atrophy and fatty degeneration of the muscles.[1]

(*c*) It is still somewhat a matter of conjecture whether cases of paralysis following typhoid, typhus, and malarial fevers are due to an affection of the peripheral nerves or of the spinal cord. The researches of Pitres and Vaillard[2] have shown that extensive degeneration of peripheral nerves is to be found in the bodies of patients who have died of typhoid fever, and they have also demonstrated that these fevers are often followed by local neuritis as a sequel. Cases of multiple neuritis with autopsies have not as yet been reported after typhoid or typhus; but Buzzard has recently recorded[3] two cases, following malarial fever, in which all the symptoms pointed to an affection of the peripheral nerves. In this country Gibney has described several cases of paralysis of the extremities, of sudden onset, rapid course, and prompt recovery under large doses of quinine, which he considered malarial. Probably the following case, which was kindly sent to me by Dr. L. E. Holt, was of this nature:

Observation XIX.—Peter S——, aged ten, a healthy boy, was suddenly seized with a chill and fever, and with pains of a severe kind in both legs, associated with weakness so that he could hardly stand. The pain was referred to the sciatic nerves, and was accompanied by numbness and tingling of the feet and legs. There was marked tenderness both in the course of the sciatics and in the muscles of the thigh and leg. There was loss of tendon reflexes, preservation of skin reflexes, and marked paresis in all the muscles of the legs, so that he required help in walking. There was no ataxia, and no incontinence of urine. The symptoms lasted about twenty-four hours, and then decreased in severity gradually during the following day, but recurred on the third, and, after a remission, on the fifth day, with lessened severity. The periodicity of the affection and the rapid cure under quinine left no doubt as to its malarial nature. During the third day, at the time when I saw him, there was redness along the course of the sciatics, and in the region of the right median nerve, and extreme tenderness along these nerve-trunks, in addition to the symptoms mentioned. The electric condition was, unfortunately, not tested; but in Westphal's case faradic contractility was totally abolished during the attack.[1] In this case the active manifestation of the malarial poisoning was by a general neuritis. In Buzzard's cases the malarial attacks preceded the nervous affection. The tenderness in muscles and nerves left no doubt regarding the peripheral nature of the disease.

---

[1] P. Grocco, Milano, 1885; Centralbl. f. Med. Wissen., 1885, p. 693.
[2] Rev. de Méd., Des Névrites périphériques, 1885, p. 980.
[3] Paralysis from Peripheral Neuritis, p. 104.

And here I may suggest that it is possible that many minor nervous affections of transient nature and of malarial origin, if more carefully examined, will be found to be due to some abnormal condition in the peripheral nerves. I allude not only to neuralgiæ, but also to visceral neuroses, whose clinical features have been so graphically described by Dr. Clifford Allbutt,[2] and to numb fingers and toes, and to hyperæsthetic and painful areas on the surface, and that host of symptoms of a local character so often carelessly ticketed neurasthenia and dismissed from consideration as soon as named. There is much in the study of the phenomena of neuritis to enforce this suggestion which cannot be developed here.

(*d*) We come, lastly, to those cases of multiple neuritis which occur in tubercular patients, or in those who have had syphilis. If a review of the cases of multiple neuritis already cited be made, it will be found that quite a number of the patients died of phthisis. This was true of the cases of Joffroy, Eisenlohr, Strümpell, Webber, and Müller. Oppenheim[3] has reported cases of multiple neuritis in tubercular patients which went on to recovery. The following case died:

---

[1] Neurol. Centralbl., 1885, p. 287.

[2] Gulstonian Lectures, 1884.

[3] Oppenheim: Zeitschr. f. Klin. Med., 1886, p. 230.

Observation XX.—A young girl, who had had syphilis, and was suffering from phthisis, took a severe cold and began to have pains in the joints, weakness, and numbness of the lower extremities. The weakness of the legs increased rapidly, and was attended by rapid diffuse atrophy. The same symptoms soon developed in the arms. Four weeks after the onset partial RD was present in all the extremities, and total paralysis of the legs and nearly complete paralysis of the arms were present. In the course of the disease, which lasted five months, there was little pain, but great tenderness of the muscles and hyperæsthesia of the skin. The sensation was diminished in feet and hands, but this was slight in comparison with the motor symptoms. The paralysis finally attacked the trunk muscles and the diaphragm, while the beginning of rapid heart action was thought to indicate paralysis of the pneumogastric nerve. Great prostration, delirium, incontinence, bed-sores, and œdema of extremities, with fever, preceded death.

Autopsy : High degree of degeneration of the peripheral nerves, including the phrenic and pneumogastric, was found, with a moderate degree of atrophy of the muscles. Cord normal, anterior motor roots normal. The changes in the nerves were similar to those after section, viz., a simple degeneration of the fibres with some increase in the connective tissue, but there were no evidences of changes in the vessel-walls, no infiltration with cells. The nerves contained very few normal fibres. The myelin sheaths were found in all stages of destruction, down to an entire absence of contents of the sheath of Schwann. Throughout the fibres fatty and granular masses were found. The sheath of Schwann did not, however, show an increase of nuclei.[1]

Such a case as this would have been ascribed, a few years ago, to a tubercular spinal meningitis, as would many of the other cases already alluded to. It is not my purpose to dispute in any way the fact that many cases of paralysis occurring in the course of phthisis are due to lesions of the central nervous system and its membranes; but I desire to emphasize the fact that a multiple neuritis may produce paralysis in tubercular individuals, and the importance of appreciating this possibility cannot be too strongly urged, since the treatment, as well as the prognosis, will differ widely according to the diagnosis made. It has been known for some time that local neuritis may complicate phthisis, and the researches of Pitres and Vaillard have recently confirmed this fact. But the complication of multiple neuritis is one not mentioned in the text-books, and yet is one which is by no means rare. It seems not unlikely that some of the nervous symptoms in consumption, formerly considered functional, may be traced to this affection.

---

[1] Vierordt: Arch. f. Psych., 1883, xiv., 3.

Although syphilis has been described as a cause of neuritis, I cannot find that the lesion in the few cases examined has had the peculiar characteristics of syphilitic lesions elsewhere in the body, and therefore it seems to me doubtful whether we are justified in describing a syphilitic multiple neuritis. Further facts are needed to establish its existence.

(*e*) It only remains to consider briefly the epidemic form of multiple neuritis, and our study of the infectious cases will be complete.

In 1882 Professor Scheube, of Tokio, Japan, called the attention of European physicians to the existence of a peculiar affection prevailing among the Japanese.[1] It was called kakke; from two Chinese words, *kiaku*, meaning legs, and *ke*, meaning disease. It had been known among the Chinese for centuries, being mentioned by name in Chinese medical books written two hundred years before the birth of Christ, and fully discussed by an eminent author in 640 A.D. It ceased, however, to prevail in China about two hundred years ago, and its ravages are now confined, according to Scheube, to Japan. There its importance is considerable, since it is so prevalent that in 1877 fourteen per cent., and in 1878 thirty-eight per cent., of the men serving in the army suffered from it. It is considered a miasmatic infectious disease by Scheube, although an eminent Japanese authority considers it due in some way to the diet of rice. That diet has something to do with its occurrence is proven by the fact, communicated to me by Dr. Wallace Taylor, that since wheat has been substituted for rice in the diet of some of the barracks and prisons in Japan the disease has been less common. It occurs in epidemics, but is always endemic in Japan. It does not attack Europeans. It affects females rarely, only nine per cent. of the cases being in women; and it is the youth of the land, between the ages of sixteen and twenty-five, who are attacked. Exposure to damp and cold in crowded dwellings, such as barracks, increases the liability to the affection. The majority of the cases occur during the hot months, but some are always under observation. This disease is not, however, confined to Japan. It has been observed for many years in the islands of the Pacific Ocean, in India, Ceylon, on the west coast of the Red Sea, in Borneo and New Guinea, in Brazil and

---

[1] Deut. Arch. f. Klin. Med., xxxi. and xxxii.

Cuba, and its prevalence in the Dutch possessions in the China Sea has made it familiar to physicians from Holland who have visited these colonies. It is there known under the name of beriberi. It is endemic in these regions, but occasionally occurs as an epidemic. An interesting account of such an epidemic, occurring in 1882–83 in Manila, the chief city of one of the Philippine Islands, has been given by Dr. Koeniger.[1] It appears that in the fall of 1882 an epidemic of cholera occurred in Manila, of such severity that twenty thousand persons, in a population of four hundred thousand, were affected. As a precaution against this disease the native population lived for several months almost exclusively upon rice, refusing to eat fruit or fish, which are their other chief articles of diet. As the epidemic was subsiding a terrible cyclone devastated the city, destroying the light wooden houses, and leaving sixty thousand families homeless; and these poor people were exposed for several weeks to the inclemency of the weather, which at this time of the year is rainy. A few days after the cyclone the epidemic of beriberi began, and as the disease had never before appeared in Manila the unknown affection excited great alarm. This was increased by its fearful mortality, sixty per cent. of the early cases proving fatal. Europeans were exempt, with two exceptions, and the Chinese population did not suffer greatly, but among the natives the epidemic was wide-spread. Thus, in one suburb of Manila, of twenty-five thousand inhabitants three hundred died in the course of eight weeks. Men and women were equally affected, and persons of all ages, except young children were attacked. The disease terminated fatally in from ten days to five weeks after its onset; but as time went on the proportion of recoveries increased, and by the end of March, 1883, it had almost disappeared. The months from October to March are the dry, cool season in the Philippine Islands, although the climate is tropical. Exposure to heat could hardly be considered a cause of this epidemic, but whether the exposure to cold and damp, or the diet of rice, or the transportation of some infectious agent by the cyclone was the cause, gave rise to much discussion, and could not be determined.

Sporadic cases of beriberi, or kakke, occasionally appear in our hospitals, usually in the persons of Chinese or Malay sailors, or in the persons of travellers from tropical climates who have been exposed to the infection in the place from which they came. Three such cases have recently been reported by Dr. Seguin, of this city,[2] in patients who came from the West Indies, and a case observed in Bellevue Hospital, by Dr. J. West Roosevelt, was discussed in the Academy of Medicine recently. In 1881 a Brazilian naval vessel entered San Francisco with a large number of the crew affected by the disease. They were sent to the United States Marine Hospital, and attended by Dr. Hebersmith, who gave an interesting account of the circumstances leading to the development of the disease in the United States Marine Hospital Report.[1] From this report the following map is taken, which demonstrates the localities in which beriberi is endemic.

---

[1] Deut. Arch. f. Klin. Med., xxxiv.

[2] Medical News, Philadelphia, December 11, 1886.

Only last year a commission was appointed by the Dutch Government to investigate the subject of its nature, and the recently published report contains the following statements :[2] The disease is caused by a micro-organism resembling the bacillus of splenic fever, though somewhat smaller, which colors with fuchsin and gentian-violet, and can be seen with a power of 560°. These bacilli are found in the blood, lungs, heart, brain, cord, and nerves of the patients, and can be cultivated outside of the body. The germs infect wooden dwellings chiefly. They may be conveyed by articles of clothing, and probably enter the body by the lungs. Direct contagion has not been observed. A potent predisposing cause to their reception in the body and to the development of the disease is lack of nutrition, consequent upon exposure to damp and to cold, and upon insufficient or bad food. It must, however, be added that a most thorough examination in Dr. Roosevelt's case, by Dr. Prudden, failed to reveal the presence of such bacilli.

In the light of these recent investigations a new view is taken of an epidemic of a peculiar kind which occurred in France in 1828. Buzzard has found an account of this, prepared by Graves, in which the symptoms are so fully detailed as to leave no doubt that it was an epidemic of multiple neuritis.[3]

The cases of beriberi are divided into two general classes, according to their severity.

There are, first, slight cases, in which the onset is gradual, being usually preceded by a little fever, coryza, and conjunctivitis, which cease when the actual symptoms commence. The patient first notices a weak and heavy feeling in his legs, and finds that he tires so easily that he cannot walk as much as usual. The tired feeling is soon associated with numbness and pain in the legs, and

---

[1] Marine Hospital Report. 1881.

[2] Deutsche Med. Woch., December 9, 1886 (an abstract only is given). See also Harada : Neurol. Centlbl., 1885, p. 326.

[3] Buzzard : On Paralysis from Peripheral Neuritis, p. 37.

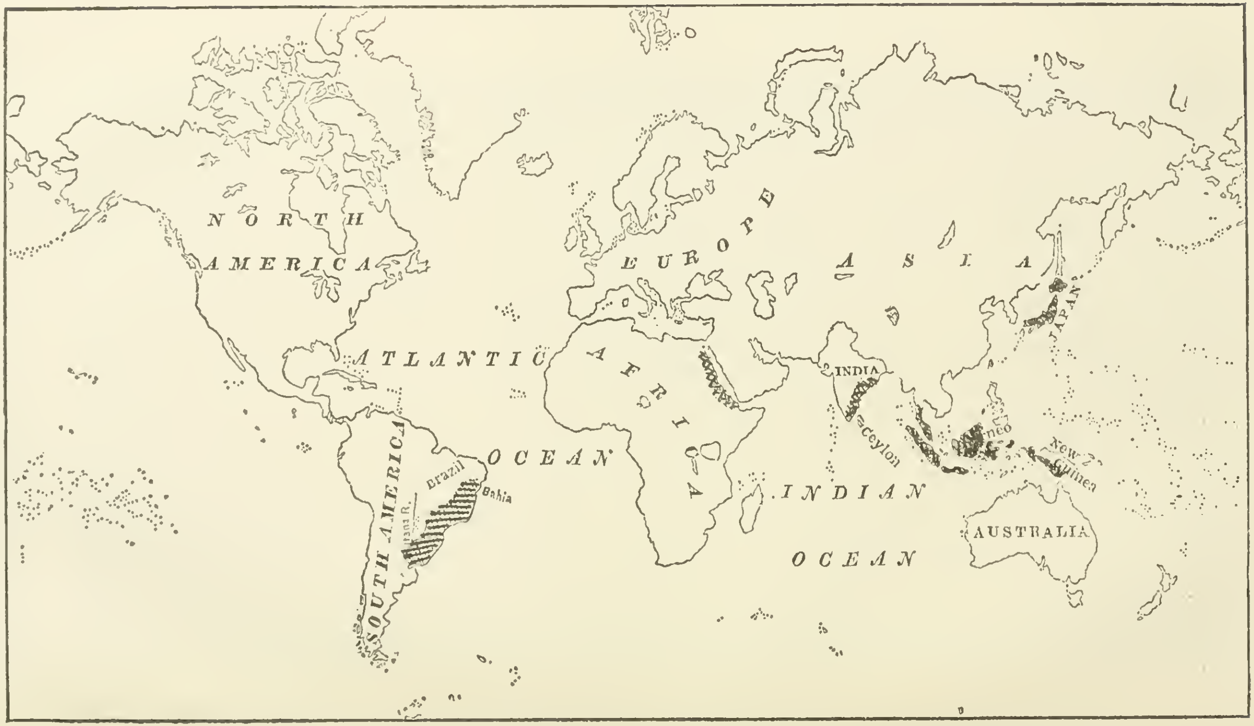

Fig. 6.—Sketch Map indicating the Geographical Distribution of Beriberi. (From United States Marine Hospital Report, 1881.)

with a slight œdematous swelling. Then, if not before, palpitation of the heart, oppression and weight in the epigastrium, loss of appetite, and general malaise are felt, and the patient finds it necessary to apply for treatment. An examination then shows some diminution of power in the feet and legs, and also in the hands, with loss of tendon reflexes, and much tenderness in the muscles, which show a diminished electric excitability. There is never any ataxia, though the patient sways when his eyes are closed. There is discovered a slight degree of anæsthesia, of irregular distribution, chiefly in the legs and in the radial-nerve region on the forearms. Though the patients look pale, it is usually impossible to find anæmia by examination of the blood.[1] The circulation in the extremities is sluggish. The heart is irregular and rather rapid, and the œdema of the extremities indicates a failure of its power. Dr. Wallace Taylor[1] finds that a sphygmographic tracing is characterized by a sudden high upstroke in ventricular systole, by a precipitous descent from the apex of the percussion-wave, and by dicrotism. Beyond this point these cases, which make up the majority, do not advance. They usually recover in a few days, or at most a month; although a few become chronic and require several months before the cure is complete.

There are, secondly, severe cases. These may present three different types. There is the atrophic or dry type, in which, after an onset similar in nature to that in the slight cases, but much more rapid, the weakness develops into a true paralysis, associated with marked wasting of the muscles and reaction of degeneration, with great diminution of galvanic excitability. Within a week the patient has to go to bed, and then the paralysis soon spreads from the legs to the arms, and may involve the trunk and even the face. The entire muscular system wastes away till the patient is a mere skeleton. In addition to the motor symptoms there is great sensory disturbance. The suffering from pain, paræsthesiæ, and general muscular tenderness is extreme, and the patient lies totally helpless and unable to tolerate the lightest touch. The skin may be glossy. There is usually some anæsthesia, but it is never complete, although it may involve the entire body. The temperature sense is seldom affected. Pain may be delayed in transmission. There are no gastric symptoms, and no œdema. Some cases prove fatal from general exhaustion, or intercurrent disease, but the majority recover after a convalescence which lasts a year or more, during which the muscular system is rebuilt.

---

[1] Wallace Taylor, M.D.: Studies in Japanese Kakke. Osaka, 1886.

There is, secondly, the hydropic or wet type. In these, heart-failure appears early, and is associated with a marked decrease of arterial tension and much œdema of the entire body, effusion into the cavities being added to that beneath the integument. The swelling of the edematous parts conceals the atrophy which is going on in the muscles, but this is indicated by the paralysis, which is as severe as in the preceding form, and it becomes evident during recovery when the œdema has subsided.

There is, thirdly, the acute pernicious type. In this all the symptoms of the two former types appear in rapid succession, and, in addition, gastro-intestinal symptoms and a suppression of urine combine to make the condition an alarming one. Effusions into the pleura and pericardium appear early. The pulse becomes small and irregular, and cyanosis indicates the heart-failure which precedes death.

In this form the disease may run its course in two weeks to a fatal termination. This was the form which chiefly prevailed in Manila, the cases of the atrophic form being the ones which recovered.

The severity differs much in different epidemics, the mortality varying from two per cent. in Japan to sixty per cent. in Manila. It is usually not above three per cent. In all the forms there is some danger of a sudden heart-failure, and this is usually the cause of death.

The morbid anatomy of the disease is, primarily, a multiple peripheral neuritis, with, secondarily, numerous organic changes in various organs, none of which, however, are essential to the disease.[1]

While we cannot join in the wish expressed by an enthusiastic German author, that the disease may soon become a familiar one to those outside of Japan, we cannot pass it by without a brief reference, especially as it resembles in so many of its features, as well as in its pathological basis, the condition which has occupied our attention at present. And it may not be unprofitable to obtain a clinical picture which differs somewhat from that already viewed, in connection with the forms more familiar to us, in order to detect sporadic cases of the disease, if such should appear among us. As to its treatment, it may be mentioned that quinine failed to influence its course, and that heart-stimulants to combat the dangerous complications, hypnotics to counteract the pain and insomnia, and general tonic treatment have proved of the greatest service. Change of climate often is attended by recovery. In the stage of recovery electricity and massage have been employed with advantage.

---

[1] Scheube: Virch. Archiv, Bd. 95, s. 146.

III.—In a considerable number of cases of multiple neuritis no cause of the disease could be definitely ascertained. Great fatigue by walking, or by hard labor, and exposure to dampness and cold are assigned as causes in some of these cases, and may have acted as etiological factors. In others the disease seems to have developed spontaneously.

The following cases are cited as belonging to this class:

Observation XXI.—A perfectly healthy man, aged thirty-four, after a long and fatiguing walk was suddenly attacked with weakness of the legs, pain, and formication, which increased so rapidly that in two days he could not walk, and in eight days was totally paralyzed—legs, trunk, arms, face, and pneumogastric nerve being all involved. The paralysis was attended by severe pains, tenderness along the nerves, profuse perspiration, fever, and jaundice. Further, a diminution of sensibility, a loss of tendon reflexes, a loss of muscular sense, a rapid atrophy, with reaction of degeneration, were observed; and trophic disturbances, consisting of an abnormal growth of hair and pigmentation of the skin, as well as vasomotor disturbances in the form of œdema, appeared. There were no fibrillary muscular contractions, no increase of mechanical excitability, and no incontinence of urine or fæces. During the following four months a gradual improvement took place, which went on to final recovery a year and a half after the onset. The only anomaly at that time was an absence of contractility of the muscles to the electro-current, when excited through the nerve.[1]

Observation XXII.—A female, aged forty-two, previously in good health, was attacked, on October 1st, with pain in the back and in all four extremities, attended by great weakness, which increased so rapidly that on October 13th there was found to be an almost total paralysis of both legs, of the trunk, and of certain muscles of the arms. The pains were accompanied by a feeling of formication and a painful sensation of tingling. When first seen by Dr. Guttmann there was total paraplegia, slight movements in fingers and toes being the only motion possible; the muscles were all relaxed and flabby, were much atrophied, were tender to pressure, and a few of the smaller muscles showed slight diminution of faradic excitability. There was no disturbance of sensation. The tendon reflexes were abolished. The sphincters were not involved. The temperature was normal, but the pulse was unduly rapid. There were no bulbar symptoms. Under large doses of iodide of potassium and the application of faradism, the patient recovered completely in the course of three months.[1]

---

[1] R. H. Pierson: Ueber Polyneuritis Acuta, Volkmann's Klin. Sam., 1883, No. 229.

In some of the cases of this form the early pains were located in the joints, and led to the diagnosis of rheumatism. The fact is merely mentioned to call attention to the possibility of mistake. A careful examination will never fail to correct it.

SYMPTOMS.—Turning now to the consideration of the individual symptoms of multiple neuritis, and their course, we are at once impressed by their number and variety.

The *sensory symptoms* are the earliest to appear and the last to pass away. In the majority of the cases on record, from whatever cause, numbness, tingling, or formication usher in the disease. These forms of paræsthesiæ begin in the feet and hands, and extend to the knees and elbows. They may be associated with burning, stretching, boring, or tearing sensations, which distress the patient, especially during the onset. But all such sensations usually subside as the affection reaches its height. Their recurrence, as the case goes on, may be regarded as a favorable symptom, however annoying, for they frequently precede recovery, and are among the last evidences of the disease to disappear. Pain is usually present as well as paræsthesiæ. It may occasionally be sharp in character ; but is usually moderate and not continuous. At times it may be lancinating, and so severe as to necessitate the use of morphia. But it is rarely as distressing as in cases of locomotor ataxia. Tenderness in the nerves and muscles is a constant symptom. It may be so marked that the limbs cannot be moved or handled, and thus it may interfere with the application of electricity and massage. When the tenderness and pain are referred to the joints, as not infrequently occurs in the early stage of the disease, the case may be mistaken for one of acute articular rheumatism, and if the joints are swollen or the limbs edematous the difficulty of diagnosis is greatly increased.

In addition to these subjective feelings some demonstrable disturbance of the various sensations is usually present. Hyperæsthesia to touch, and also to electricity, is not infrequently observed during the first few weeks. It is usually followed by some anæsthesia, although this rarely becomes complete. In some cases the loss of tactile sense is quite evident from the outset, either limited to the cutaneous distribution of some special nerve, in which case oddly shaped areas of insensibility will be found, or, as is most often the case, about uniformly distributed over the distal parts of the extremities. When the anæsthesia is at its height the patient has difficulty in locating a touch upon the limb, even though he feels it. The transmission of pain and temperature-sensations is always delayed, but the impressions are usually felt quite acutely. The sense of pressure has been tested in only a few cases, and in those it was decidedly impaired. The muscular sense escapes any affection in some cases, but in others is the most profoundly disturbed of all the senses. When it is involved, the inco-ordination and ataxia are well-marked symptoms, and, as already stated, some of the cases have been mistaken for locomotor ataxia, because of the predominance of the disturbance of muscular sense.

---

[1] Guttmann: Deut. Med. Wochenschr., 1884, No. 19

These sensory symptoms are usually limited to the forearms and hands, and to the legs and feet. In a few cases they have involved the entire extremities, and even the trunk; and one case of facial tingling, with anæsthesia, has been recorded.[1] The skin reflexes are usually preserved.

The special senses are rarely affected in multiple neuritis. It is true that optic neuritis has occurred in a few cases, and in two cases hearing as well as sight has been affected. These cases prove that no nerve can be said to be exempt from implication in this disease, but the liability to affection seems to be slight in the case of the nerves of special sense.

The *motor symptoms* are as marked and as important as the sensory. Paralysis, beginning as simple weakness, with a feeling of fatigue on any exertion, gradually increases in severity until at the height of the disease it becomes complete. It usually comes on rapidly, so that within two weeks the patient is helpless; but it may be less sudden, and not deprive him of the power of walking and of using his hands for two or three months. In a few cases a very acute onset is recorded, all the symptoms developing within three or four days. The distribution of the paralysis is not uniform at the outset. It may develop in the muscles supplied by a single nerve, and advance to others; it may begin in all the muscles of the legs, and then involve those of the forearms; it may commence in all four extremities at once. It is always more severe in the muscles which move the joints of the feet and hands, and the ankles and wrists. It rarely invades those which move the knees and elbows. When the disease is fully developed, all the muscles below the knees and elbows are much weakened or totally paralyzed.

---

[1] Lowenfeld: Neuro. Centralbl., 1885, No. 7.

In a few cases those of the thighs and arms are involved also, and occasionally the muscles of the trunk and those of respiration become affected and the patient dies. When such a paralysis makes rapid progress and involves all the body, the case resembles Laudry's paralysis. But in multiple neuritis, as has been already stated, the disease does not creep up from legs to thighs, and then to trunk and arms, as in Laudry's paralysis. It spreads from feet to hands, from legs to forearms, and the trunk is invaded only at the end, and in Laudry's paralysis sensory symptoms are wanting. In some cases of multiple neuritis the cranial nerves become involved; those of the eye and of the face being most liable to invasion. It is only in fatal cases that the action of deglutition has been affected; and when the pneumogastric is invaded, and the heart becomes rapid and irregular, the prognosis is always grave. Spasms rarely occur.

The paralyzed muscles are relaxed, flabby, and atrophied; they may or may not lose their mechanical irritability, but their normal tone is always lost, and hence the so-called tendon reflexes are abolished.[1] To the electric current their excitability is very rapidly and markedly changed; but the conditions which have been observed are quite various. Sometimes there is a simple diminution of excitability, and then a very strong faradic or galvanic current is needed to produce contractions. Frequently all faradic excitability is lost, and then the muscles react to a galvanic current only. In this condition it may require a very strong galvanic current to produce contraction, and this fact is quite pathognomonic of neuritis. For an anterior polio-myelitis, where the muscles respond to galvanism only, it does not require a strong current to cause a motion until some months after the invasion. The action of the different poles is not uniform. In many cases the contraction of the muscle when stimulated with the positive pole is greater than when stimulated with the negative pole, and the contractions may be sluggish. Then the reaction of degeneration is present. But in some cases the normal condition is found, and the negative pole produces stronger contractions than the positive pole. If the muscles that are not paralyzed be tested, the same electrical changes may often be discovered in them. A loss of faradic irritability and a marked decrease in the galvanic irritability of the muscle and nerve are, therefore, important symptoms of multiple neuritis. And as the disease goes on to recovery a gradual increase in the galvanic irritability occurs, a fact which is often of much aid in prognosis if careful measurements of the strength of current used be made by the galvanometer. I am accustomed to record such measurements upon charts, and thus to obtain an electric curve for each muscle which is paralyzed.[1] These curves enable one to judge of the progress of the case quite accurately, and when the line is advancing steadily toward the normal point, after a great deflection or after a stationary level, the prognosis is very favorable.

---

[1] A few exceptions to this rule are found in two cases recorded by Strümpell and Mobius, Munchener Wochenschrift, 1886, No. 34, and in Brit. Med. Jour., January 1, 1887.

As a result and accompaniment of the paralysis *abnormal positions* are assumed by the limbs. The dropped wrist and dropped foot are quite characteristic of multiple neuritis. But other deformities may be present. In a few cases there have been extreme contractures of all the extremities in flexed position. And not infrequently the appearance of the claw-shaped hand, and some one of the various forms of talipes, indicates a serious shortening of one set of muscles, and corresponding weakness of its opponents. These deformities usually subside as the power returns, or if they do not they can be corrected by proper manipulation and by apparatus. In a few cases it has been necessary to resort to tenotomy, but a permanent deformity has not been recorded.

The *vaso-motor and trophic symptoms* are less constant than those already described. In some cases marked œdema has been an early and a permanent symptom. This may develop in the feet and hands, or may appear about the joints. It usually is temporary. The circulation is not impaired any more than is customary in a limb whose muscles are inactive, and coldness and cyanosis are rarely sufficient to attract attention. Sometimes profuse perspiration is a noticeable symptom, being limited to the paralyzed parts. It may be offensive, and by its evaporation always causes a complaint of coldness. In other cases glossy skin makes its appearance early, and remains until regeneration of the nerves begins. Its disappearance in one of my own cases was the first sign of recovery in the lower extremities. Other forms of trophic disturbance are rarely met with in multiple neuritis. And this is quite remarkable in view of the fact that it has been the tendency of late to refer such trophic affections as ulcerations, bed-sores, gangrene, pemphigus, and various eruptions to lesions of the nerves.[2] It is true that inflammations of the joints, resembling those appearing in acute rheumatism, sometimes occur at the onset of neuritis ; but as they disappear quickly, while other symptoms remain, it is improbable that they are to be traced to the changes in the nerves. They may be due to the infectious agent, or to the same obscure cause which sets up the neuritis, or they may be evidence of an attack of acute articular rheumatism, which is in turn followed by neuritis ; but they cannot be described as trophic symptoms of the disease, otherwise they would be more constant in their occurrence and more permanent in their duration.

---

[1] Electric Charts, Journal of Nervous and Mental Diseases, February. 1887.

[2] Raynaud's disease has recently been ascribed to neuritis. See J. Wiglesworth, Brit. Med. Jour., January 8, 1887.

A negative symptom of some importance is the absence of any interference with the automatic acts controlled by the sphincters.

One word about the onset. Occasionally it is sudden and accompanied by a marked febrile movement, with chill, and temperature of 103° to 104.5° F. The fever may persist for several days, but usually subsides spontaneously and does not recur. In a few cases there has been a constant elevation of temperature from one-half a degree to one and a half degree above the normal ; and an increase in the rapidity of the pulse throughout the disease has been noticed. A pulse of ninety need give no alarm, but if it runs up to one hundred and forty, and becomes irregular, there is reason to believe that the disease has attacked the vagus nerve, and then the prognosis becomes serious, though not by any means hopeless.

The duration of the disease varies considerably in different cases. An average of twenty-five cases gives seven months as the probable time required for complete recovery. But in these cases the duration varied from two months, in the most favorable, to sixteen months, in the most refractory.

The disease is more common in males than in females, excepting the form produced by alcoholism. Of 47 non-alcoholic cases, 33 were males.

All ages are liable to be affected, but in the records of multiple neuritis, excepting the form produced by diphtheritic poison, the cases recorded do not include any children.

That children may be affected is, however, not at all improbable. Dr. H. D. Chapin, of this city, has described four[1] cases of atrophic paralysis in children, in which the presence of sensory symptoms, pain and muscular tenderness, and the steady progress toward recovery pointed to the existence of multiple neuritis. The cases were such as are usually called infantile paralysis, but there were points of difference (viz., the existence of sensory symptoms) which removed them from this category. Leyden called attention to the possibility of mistaking neuritis for poliomyelitis in his first article upon the disease, but he referred to adult cases rather than to children's cases. And if the surmise of Chapin should be substantiated by autopsy, it might be necessary to conclude that in children, as well as in adults, the majority of cases of supposed poliomyelitis which recover are cases of multiple neuritis.

---

[1] Painful Paralysis in Children, NEW YORK MEDICAL RECORD, January 15, 1887.

In adults, out of 50 cases 6 were between the ages of 18 and 20; 8 between 20 and 30; 20 between 30 and 40; 8 between 40 and 50; 5 between 50 and 60 and 3 over 60 years of age.

DIAGNOSIS.—While the individual symptoms occurring in the course of multiple neuritis are not different in character from those found in spinal-cord diseases, the diagnosis can usually be reached with very little difficulty when their combination, the causation, and the course of the case under examination are considered. The varying combination of the symptoms possible has been manifest during their description, and it is not my intention to review them again. There are, however, three combinations which resemble very closely, respectively, anterior poliomyelitis, locomotor ataxia, and diffuse myelitis, and to these attention must be directed.

Atrophic paralysis, with reaction of degeneration and loss of reflex is common to anterior poliomyelitis and some cases of multiple neuritis. In the latter a more gradual onset, preceded and attended by numbness and pain, tenderness in the course of the nerves, tenderness in the muscles, and the persistence of sensory symptoms after the invasion, will remove all doubt regarding the diagnosis. When these symptoms are not clearly marked, the distribution of the paralysis in symmetrically situated muscles, especially if these muscles are supplied by single nerves, and the further extension to muscles in other nerve domains, rather than the affection simultaneously of muscles which are grouped physiologically (*i.e.*, act together to perform one function), will point to neuritis. In neuritis the paralysis advances more or less gradually, while in acute poliomyelitis there is, after the onset, a subsidence of the paralysis in some of the muscles first involved. And lastly, as the case goes on, a gradual complete recovery will be far more frequent if it was originally a case of multiple neuritis.

Ataxia, loss of knee-jerk, pain, and sensory disturbances, including a loss of muscular sense, Romberg's symptom, and optic neuritis are common to locomotor ataxia and multiple neuritis. In the latter the relatively rapid onset of the ataxia, which follows closely upon the sensory symptoms; the prominence of numbness and anæsthesia, rather than of lightning pains; the extreme degree of the anæsthesia and analgesia; the tenderness of muscles and nerves; the usual occurrence of some degree of actual paresis with atrophy and RD; and the absence of bladder and sexual symptoms will point inevitably to the diagnosis. Furthermore, the ataxic form of neuritis only occurs after poisoning with alcohol or arsenic, or as a sequel of diphtheria, and the establishing of the causation will aid the diagnosis. Here, again, the course of the case toward recovery, with return of the knee-jerk, will decide in favor of neuritis, if the diagnosis has not been reached in an early stage.

There are very few symptoms of diffuse myelitis which are not found in cases of neuritis. But cases of diffuse myelitis of the type of Duchenne—paralysie générale spinale subaigüe ascendante—are very rare, and indeed by Leyden it has been affirmed that all such cases are multiple neuritis. Other authorities dispute this assertion and leave us to establish points of difference. These are as follows: In neuritis affections of the functions of micturition and defecation do not occur. Girdle sensation is very rarely mentioned as a symptom; bed-sores and cystitis have not been observed. The advance of the paralysis is rarely from the legs to the thighs and trunk, and then to the arms; it is usually from the legs to the forearms, the trunk and thighs escaping, and, as a rule, the distal portions only of the extremities are paralyzed. If the muscles of the abdomen and respiration are involved, it is only in rapidly fatal cases. In neuritis there is usually some ataxia, and loss of muscular sense is quite evident; while in some, at least, of the cases of myelitis of Duchenne, there were no sensory symptoms at all. Finally, the tenderness of muscles and nerves, and the absence of tenderness to pressure or to heat in the spine, would decide in favor of neuritis. The diagnosis from meningitis of the cord, from tumors or hemorrhages into the cord, or from general paralysis of the insane, would rarely present any difficulty to one who was familiar with the symptoms in those affections, and who knew the prominent features of multiple neuritis.

No small difficulty may be encountered, however, in settling the question whether in a given case we have to deal with multiple neuritis alone, or with multiple neuritis which is complicated by myelitis. The importance of the question is evident, since the prognosis in the two conditions is very different; and the number of autopsies on record in which this complication has been demonstrated, though few, is sufficient to make a decision of the question necessary.[1] The following points may enable a determination of the question to be reached: (1) As long as a case is increasing in severity, or in the extent of the symptoms, no one can determine the extent of the lesion. It is only when its course has become stationary that the question of exact limitation will arise. Many cases remain practically without improvement for three or four months, and then gradually recover. A stationary condition alone does not, therefore, excite fear of a complication. But neuritis tends spontaneously to recover, the process of regeneration beginning soon after the degeneration has ceased. If, therefore, there appears to be no improvement of the condition after the fourth month, the probability is either that the cause of the disease has not been removed, or that a myelitis has developed and prevents recovery. (2) The course of a case of neuritis is quite characteristic, the symptoms reaching their maximum in a short time, and then subsiding. If, after a stationary period, further symptoms develop, we must believe either that the cause of the disease is renewed (*e.g.*, the use of alcohol) or that myelitis has begun. The symptoms of such a myelitis will be an increasing weakness and more rapid and progressive atrophy of the muscles; a gradually decreasing degree of galvanic excitability in the paralyzed muscles; a loss of pain and temperature senses, which, as a rule, are not affected in neuritis; a decrease in the paræsthesiæ, and an increase in the degree of anæsthesia; the development of loss of control over the sphincters; the occurrence of bed-sores, furuncles, eruptions of a bullous nature; and the beginning of cystitis. (3) On the other hand, if the symptoms are gradually improving; if the power gradually returns; if the anæsthesia decreases, and is succeeded by paræsthesiæ, however disagreeable to the patient; if the galvanic excitability becomes gradually more acute in the muscles, so that the electric curve approaches the normal line; if the faradic contractility returns in the muscles; if the tenderness of muscles and nerves decreases; and if the glossy appearance of the skin disappears, it may be stated that no complication of myelitis has occurred, and that recovery, though possibly prolonged, will at length result.

Another question of diagnosis must also be considered. It is found that in no small number of cases of locomotor ataxia multiple neuritis develops as a complication. How can we determine when this complication exists? The symptoms of the two diseases may be so nearly identical that difficulty arises in distinguishing them. When they coincide it is equally difficult to determine to which disease any given symptom belongs. Pitres and Vaillard,[1] who have considered this subject most carefully, affirm that there is no constant relation between the severity of the central and peripheral lesions when they coincide. But there are certain symptoms which develop in some cases of tabes, but not constantly, which may be looked upon as accidental, and thus traced to neuritis. Such are the appearance of plaques of anæsthesia and analgesia of limited area, muscular weakness or paralysis of limited extent, trophic disturbances in the skin (*e.g.*, perforating ulcer), nails, joints (*e.g.*, Charcot's disease), bones (*e.g.*, spontaneous fractures), and teeth (*e.g.*, spontaneous falling out), and possibly the various crises referable to the viscera and larynx. In cases which competent observers have examined, peripheral neuritis has been found *post-mortem* in the nerves supplying the parts in which these symptoms appeared. It is, therefore, reasonable to conclude that in any case of locomotor ataxia in which these symptoms develop we have to deal with a posterior sclerosis which is complicated by a peripheral neuritis. And here again the distinction has a bearing upon the prognosis, for the symptoms of the accidental kind may pass off, while those due to the central lesion will remain. While these conclusions of the French authors are of importance, it must be noticed that their claim that trophic disturbances are due to a complicating neuritis is by no means substantiated by the history of cases of multiple neuritis, in which, as we have seen, trophic disturbances of the varieties mentioned do not often occur.

---

[1] Oppenheim, Zeit. klin. Med., xi., 232; Leyden, Charité Annalen, 1878; Duménil, Gaz. Heb., 1861.

PROGNOSIS.—The prognosis in multiple neuritis is good, provided the exciting cause can be removed. The only cases which form an exception to the rule are those whose constitutions are much impaired by excesses or by other diseases; those who have so far indulged in alcohol or are so completely soaked with arsenic or lead as to be unable to throw off the poison; and those in whom the disease begins with great suddenness, advances rapidly, and involves the phrenic and pneumogastric nerves. These cases either die of respiratory paralysis or of some complication. When a case has reached the stationary period the prognosis is generally favorable, and if the encouraging signs of recovery already mentioned begin to appear a cure may be promised. The possibility of the complication of myelitis must not, however, be overlooked, and if it occurs the prognosis becomes at once unfavorable. Even in serious cases of alcoholism, with gastro-intestinal and cerebral symptoms, it the acute stage is safely passed and all alcohol be removed from the patient's diet, recovery from very extensive paralysis will occur. The cases of diphtheritic paralysis and ataxia usually recover without treatment. The cases occurring with syphilis and tuberculosis are not so uniformly favorable, although the former usually respond promptly to specific treatment, and even the latter may recover wholly. In epidemics, the percentage of mortality has been found to vary, but is usually low.

---

[1] Pitres et Vaillard: Des névrites périphériques chez les tabetiques, Rev. de Méd., 1886, p. 574.

Treatment.—The treatment of multiple neuritis requires patience, but receives the reward of success. As we have already seen, the majority of the patients recover, and it is probable that, if the cause of the affection were removed and the patients placed in favorable circumstances, expectant treatment would alone be sufficient. It is, however, not advisable to let therapeutics play a passive part. The course of the disease can be altered and its duration much shortened by active interference. In the stage of invasion the free use of salicin, salicylic acid, or the salicylate of soda seems to have important results. These remedies cannot be said to act as promptly as in cases of acute articular rheumatism, but the consensus of opinion is that their effect in multiple neuritis is very marked. They should be given, as in acute rheumatic fever, in large doses until noticeable effects are obtained. They should be combined with the bromide of potash or soda, partly because these drugs counteract unfavorable symptoms produced by the salicin compounds, and partly because in the hyperæsthetic irritable condition attendant upon the invasion of the disease they are indicated. This condition may require stronger sedatives, and not infrequently morphine must be employed to give relief from the excruciating pains. The pains are often relieved by hot or cold applications to the limbs; but as the muscles are often exceedingly tender ordinary applications cannot be made. It is then advisable to use evaporating lotions, preferably those containing chloroform, which may be soaked into light cambric or gauze, and gently placed upon the limbs, which lie upon the softest pillows, or which may be more comfortable if the patient is put upon a water-bed. Applications of a five per cent. solution of carbolic acid have also been of use. If cool applications prove intolerable, heat may be employed. The limbs may be enveloped with cotton and covered with oil-silk, a light bandage keeping these in place; or they may be frequently bathed in hot water, and hot bottles placed against them, some soft substance intervening. One of my patients found great relief from the paræsthesia by cold douches, while another preferred the use of hot water. It is best to let the patient decide as long as the application has to be made for the relief of pain. Gentle friction with oil of cocoanut often affords comfort. In the chronic stage, as we shall see presently, heat is to be preferred to cold. Cases which are distinctly syphilitic, if such occur, should be treated from the outset with inunctions of mercury and large doses of iodide of potash. I believe that both these drugs should be employed together, even in the tertiary stage of syphilis, and it is my experience that all syphilitic nervous lesions, whether central or peripheral, yield more promptly to their combined use than to the employment of either alone. Malarial cases must be treated with quinine or Warburg's tincture. In non-malarial cases quinine has proved of no avail. In cases which are due to poisoning of any kind the first necessity is to eliminate the toxic agent from the system, and the second to prevent any further ingestion of the poison. Iodide of potash aids in the elimination. The second indication is easily fulfilled when arsenic or lead are the toxic agents, but when the case is due to chronic alcoholism special precautions are needed. Alcoholic cases require from the outset special treatment. The condition at the time of the onset of the paralysis may be one verging upon delirium tremens. If all alcohol is suddenly removed, without due care to supply some other heart-stimulant and to secure the perfect nutrition of the patient, serious collapse may ensue. The first necessity is therefore to take care of the general condition of the patient. If this will admit of the immediate withdrawal of all alcoholic stimulation it should be done; if not, the alcoholic beverage must be immediately reduced in quantity, and as soon as possible wholly cut off. The use of milk diet, or kumyss, or pancreatized milk, or, if necessary, rectal alimentation will be followed by a gradual recovery of the power of assimilation, and as soon as the patient ceases to lose weight all alcohol may in any case be safely stopped; its elimination by the intestines and kidneys may be hastened by appropriate means, and cerebral symptoms, if they arise, may be treated as in other cases of alcoholic intoxication. But it is in the chronic stage, when the patient is gradually recovering, that the vigilance of the physician is called into play to prevent a renewal of the poisoning. It is amazing that patients who know perfectly the injurious effect of alcohol upon them should insist upon getting it. But it is done. And when these patients are surrounded, as is often the case, by sympathizing friends, or servile domestics, or unscrupulous nurses, who do not appreciate the importance of total abstinence either for themselves or for the patient, they often succeed in baffling all attempts to deprive them of the favorite drink. It is only when they are watched constantly by persons who can be implicitly trusted, and who have sufficient authority to cut off all surreptitious supplies that the physician can feel sure that his commands are obeyed. And this precaution is by no means needless even when it is probable that family servants are trustworthy; for the continued pleading and remonstrance of the patients may corrupt the best of attendants, especially if accompanied by threats of discharge at a future day. It is therefore necessary to place these patients under the surveillance of trained nurses from the start, or to remove them to an institution where they are under control.

In the chronic stage the drugs which are of greatest service are strychnia and arsenic. Strychnia may be given in doses of $\frac{1}{60}$ to $\frac{1}{20}$ gr. t.i.d., and it is well to combine it with phosphoric acid and the syrup of the hypophosphites. Arsenic may be used in tablets or pills containing $\frac{1}{50}$ to $\frac{1}{30}$ gr. t.i.d., or in Fowler's solution, five to eight drops t.i.d. The use of iron with these two drugs will be indicated in the majority of cases where there is attendant anæmia. In alcoholic cases both arsenic and strychnia may increase the mental irritability, but should be continued unless this becomes too great. I have seen benefit from both of these drugs, and think it well to employ them alternately, using each for about two weeks at a time.

The remedies used in the chronic stage have two objects: one is to increase the rate of repair in the nerves; the other is to keep the nutrition of the muscles as good as possible. While the drugs mentioned probably meet the first indication there are other remedies which meet both. These are massage, warm baths, and electricity.

The proper manipulation of the limb increases the circulation in it. The increase of circulation brings fresh supplies of material to the nerve which is undergoing repair; it also aids the nutrition of the muscle, which would otherwise be decidedly affected by the sluggish flow of venous blood, due to the lack of functional activity. As soon, therefore, as the active progress of the disease is checked and the muscular tenderness has sufficiently subsided to allow the limbs to be rubbed, this potent remedy should be employed daily.

Allusion has already been made to baths and douches in the early stage, for the purpose of quieting sensory symptoms. In the chronic stage the object is a different one. Like massage, warm baths and douches stimulate the circulation and aid the nutrition and reparative processes in progress. Hence they are to be used daily.

And if the warm bath be given at night it will secure not only a local action but produce a general sedative effect, insuring quiet rest. In multiple neuritis as well as in many other nervous affections not due to anæmia, a warm bath at night or a warm douche to the neck and spine is far preferable to, and more efficacious than, the majority of hypnotic drugs in causing a good night's sleep.

The last agent to be mentioned is electricity. It is, however, among the first in importance, and in its use it is necessary to know what object is sought in its application. There is first the object of increasing the progress of nerve regeneration. This may be attained by the application of a constant galvanic current to the degenerated nerve, passing the current through the nerve in either direction, or in both alternately. A mild current should be employed, its strength being measured by a galvanometer. The strength of the current will depend: (*a*) on the size of the sponges placed upon the skin, (*b*) on the pressure upon the sponges, (*c*) on the resistance of the skin, and (*d*) on the number of cells of the battery used. If the sponges are two inches in diameter, six milliampères is enough; if they are three by five inches in measurement, twenty milliampères should not be exceeded. If no galvanometer is used the strength of the current employed is uncertain. But it is to be remembered that the current grows stronger the longer it passes, since the skin resistance is gradually overcome; and therefore, if the strength is measured in cells, the number of cells used should be decreased gradually during the application. As so few practitioners use a galvanometer it may be well to state that with large sponges (*i.e.*, three by five inches) wet with warm water a freshly filled bichromate of potash battery will give nearly one milliampère of strength for every cell used during the first three minutes, provided the sponges be put on any part of the body except the soles of the feet or the palms of the hands, and pressed firmly upon the skin. After the first three minutes the body resistance decreases, so that when twenty cells are used to start with, one should be cut off every half minute until the number is reduced one-third. The duration of the application should be about ten minutes to each limb. During this time the distal sponge should be passed over various parts, so as to include all the nerve-branches in the current; the central sponge should be put over the nerve-trunk high up on the limb. The current should be begun and stopped gradually, and never suddenly broken; applications may be made daily.

The second object to be obtained is to re-establish the conduction of impulses in the regenerated nerve. This is secured by the method just described. It may be attained by the use of faradism, the interrupted secondary current being sent along the nerves by placing one pole over the nerve-trunk and passing the other over the skin of the limb. The strength used should be just sufficient to be felt distinctly through the palms of the operator's hands. The third object sought is to maintain the nutrition and function of the muscles by exercising them, and thus preserving their normal irritability. This cannot be done by a faradic current as long as they do not contract to it. The galvanic current must therefore be employed. But now it is not a steady current which is needed, for this does not cause any motion. It is only when the steady current is suddenly broken and renewed that the contraction occurs. Hence place one pole over the trunk of the nerve and the other upon the muscle, and with an interrupting electrode make and break the current at the pole which is on the muscle. The pole which produces a contraction with the least current possible is the one to be applied to the muscle. This is in RD the positive, in normal conditions the negative pole. Each muscle should be exercised for three or four minutes every other day. When electrical treatment is thus employed very marked improvement is observed, which can be measured accurately if a galvanometer is used, since every week will show a change of the strength of current needed to produce muscular contractions toward the normal.

The treatment must be kept up, in the chronic stage, until recovery is complete.

If contractures have occurred in the paralyzed limbs persistent massage may overcome them. If it does not, they are to be treated on general surgical principles.

BIBLIOGRAPHY.

In addition to references cited in the text the following are important:

Joffroy, A.: Archives de Physiol. Norm. et Path., 1879, pp. 172–198.
Pitres: Archives de Neurologie, 1882, v., vi.
Tessier: Lyon Médical, 1879, xxxii., p. 417.
Roger: L'Éncéphale, 1885, No. 2.
Pitres et Vaillard: Revue de Médecine, 1885, p. 987; 1886, p. 574; and Arch. de Neurol., 1886 and 1883.
Gombault: Progrès Médical, 1886, No. 23, and Arch. de Neurol., i., 1880.
Eloy: Union Médicale, 1886, September.
Brissac: Comptes-rendus de l'Acad. des Sci., tm. cii., p. 439.
Charcot: Gazette des Hôpitaux, 1884, August 28.
Dejerine: Archives de Physiol., 1878; 1883, p. 72; 1884, p. 230.
Francotte: Rev. de Médecine, 1886, No. 5.
Caspari: Zeitschr. f. Klin. Med., v., 537.
Strube: Berliner Dissert. über Multiple Neuritis, 1881.
Oppenheim: Deut. Arch. f. Klin. Med., xxxvi., p. 563.
Kast: Arch. f. Psych., xii., p. 266.

Roth: Correspond. Blatt. f. Schweitz. Aerzte, 1883, July 1
Hiller: Berliner Klin. Wochenschr., 1881, p. 605.
Dubois: Corresp. Bl. f. Schweitz. Aerzte, 1883, No. 18.
Lowenfeld: Arch. f. Psych., xv., 1884; also, Aerzt. Intell. Bl., 1885, No. 6; also, Neurol. Cent., 1885, p. 149.
Hirt: Neurol. Centralbl., 1884, p. 481.
Erb: Neurol. Centralbl., 1883, p. 481.
Strümpell: Neurol. Centralbl., 1884, p. 241.
Krüche: Deut. Mediz. Zeit., 1884, p. 229.
Remak: Centralbl. für Augenheilkunde, 1886, June.
Verhandlungen des Congresses f. Innere Med., 1884. Leyden: Ueber Poliomyelitis und Neuritis.
Remak: Neurol. Central., 1885, p. 313.
Schultz: Neurol. Central., 1885, p. 433.
Eulan: Berl. Klin. Woch., 1886, p. 95.
Hösslin: Münch. Med. Woch., 1886, p. 41.
Freud: Wiener Med. Woch., 1886, p. 167.
Homen: Centralbl. f. Nervenheilk., 1885, p. 313.
Rosenbach: Centralbl. f. Nervenheilk., 1885, p. 375.
Broeck: Centralbl. f. Nervenheilk., 1885, p. 310.
Strümpell und Möbius: Münch. Med. Woch., 1886, No. 34.
Bernhardt: Zeitsch. f. Klin. Med., 1886, p. 363, with bibliography of alcoholic paralysis.
Oppenheim: Zeitsch. f. Klin. Med., 1886, p. 860.
Guttmann: Deut. Med. Wochen., 1884, No. 19.
Kauders: Wien. Med. Woch., 1885, No. 52.
Gowers: Diseases of the Nervous System, vol. i., p. 91.
Lancet, 1886, ii., p. 380; 1887, i., p. 28.
British Medical Journal, 1886, ii., p. 977; 1887, p. 1.
Ross: Medical Chronicle, 1887, January.
Dana, C. L.: Brain, January, 1887, with bibliography of arsenical paralysis.
Brissaud: Des Paralysies Toxiques, with general bibliography. Paris, 1886.

PROCEEDINGS

OF THE

NEW YORK PATHOLOGICAL SOCIETY.

Stated Meeting, January 26, 1887.

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

THE PRESIDENT announced, as

THE COMMITTEE ON MICROSCOPY

for the ensuing year, Drs. G. C. Freeborn, W. H. Porter, and J. S. Thatcher.

DR. W. H. PORTER presented

A HEART WITH A DOUBLE MITRAL ORIFICE,

probably congenital, but he had been unable to find mention of this peculiar malformation.

There were two other interesting features in the case. The man was admitted into the Chambers Street Hospital, where the diagnosis of uræmia was made, and he was transferred to Bellevue Hospital, where it was recorded that he had considerable serous and blood-stained discharge from both ears, and the diagnosis of

FRACTURE AT THE BASE OF THE SKULL

was made, although there was no paralysis. Death occurred a few hours afterward. The autopsy did not reveal any fracture whatever of the skull.

The other interesting fact was that the man had

A DEEP URETHRAL STRICTURE,

which illustrated one reason for not treating these strictures by internal urethrotomy; that is, the stricture was upon the *floor* of the urethra; the roof of the urethra was normal.

DR. PORTER also presented a microscopic section, made from the cortex of the brain, that showed

A SINGLE MILIARY ANEURISM

very distinctly; the globular swelling involved the whole diameter of the little vessel.

CHRONIC PACHYMENINGITIS—ACUTE LEPTOMENINGITIS.

DR. L. EMMETT HOLT presented specimens taken from a female child, twenty months old, with the following history:

The patient was one of a pair of twins, the mother and the other twin having died of tuberculosis. She had always been delicate, and in August, 1886, had a febrile attack, lasting nearly two weeks, with a temperature ranging from 102° to 105° F., for which no explanation could be found. There was a moderate amount of albumen in the urine, but no casts. The fever gradually subsided, not being influenced in its course especially by any of the agents employed. There were no marked cerebral symptoms. Ever since that time the child had remained in a rather dull, listless condition, but not particularly drowsy, and gave no signs of pains in the head or elsewhere. She did not thrive, her weight remained stationary, and her tissues were soft and flabby. She was frequently examined with the idea of the possible development of tuberculosis, but no signs could be found in the lungs to indicate disease of any character. Only two prominent symptoms were present—an irregular febrile movement and an enlarged spleen. The evening temperature generally touched only 100° or 101° F., but every week or two a sudden elevation to 104° F. would occur without assignable cause. This was true of the three months preceding her death. The spleen could easily be felt below the ribs.

Quinine was tried in full doses without benefit; then mercurials, by inunction, and later by the stomach, with the idea of a possible syphilitic element in the case. Improvement seemed to follow this plan for a time, and the enlargement of the spleen sensibly diminished, but these changes for the better were but temporary.

Four days before death very marked cerebral symptoms developed. The temperature rose steadily from 101° to 104°, then to 105½°, and finally, on the day before death, to 108° F.

The respirations became irregular, shallow, and finally typically Cheyne-Stokes; pupils equally contracted; slight rigidity of the muscles of the upper and lower extremities, but no paralysis; abdomen tympanitic; stupor increasing to coma, and then death without convulsions, either local or general, and with no focal symptoms at any time during life.

Autopsy, forty-eight hours after death.—Body small, poorly nourished; fontanelle open, $\frac{3}{4} \times 1$ inch in size. Head: Pretty strong adhesions between dura and pia over frontal lobes, especially of right side. Inner surface of dura over about one-half its extent, and most abundant near the median line at convexity, covered with jelly-like lymph of a yellowish-green color, fully one-fourth inch thick. Beneath this recent deposit a pretty tough false membrane was present, which again was separated from the dura by a thin clot partially organized, but not completely discolored, and covering an area about half the size of one's hand. The false membrane could be demonstrated as far as midway between the sagittal suture to the base of the skull. Over the left side of the convexity a very thin false membrane existed, not nearly so extensive, but no clot or recent lymph was present. The superior longitudinal sinus contained a firmly organized thrombus, which was not adherent. The pia over the whole convexity, and over the base to a less degree, was covered with recent lymph and pus, very abundant everywhere, giving the brain a greenish appearance. The brain-substance to the naked eye showed no changes; the ventricles were empty, and no tubercles were seen anywhere. Chest: Lungs and heart normal. Spleen: Weight one and one-eighth ounce; firmer than usual; size about normal. Liver normal. Kidneys showed marked cloudy swelling both to naked eye and to the microscope, but no further changes; weight three ounces. Intestines showed old thickening of the mucous membrane, but no evidence of recent inflammation. No tubercles in any viscus.

The case was of interest chiefly from the clinical history and the indefiniteness of the symptoms until a few days before death.

No symptoms were observed during life which corresponded to the meningeal hemorrhage that was found at the autopsy.

Dr. W. R. Pryor presented the fundus of a uterus, to which was attached

A TUMOR FORMED BY PYO-SALPINX.

It was removed on the 11th inst. by Dr. H. Marion Sims from a woman who had been suffering from the symptoms belonging to pelvic cellulitis. The growth was sessile, and necessitated amputation at the internal os. Patient died of peritonitis, the result of constriction from old bands, two and a half days after the operation.

There were several pus-cavities in the tumor; one was nearly ready to rupture through the peritoneal covering. The largest cavity was, probably, connected with the Fallopian tube, although it had nothing whatever to do with the mucous lining of the tube. It was a cavity filled with pus. Under proper precautions some of the contents of this cavity were withdrawn for experimental study, with reference to bacteria, and were submitted to Dr. Prudden.

The President said that the cleanliness with which the removal of the fluid was done was proven by the fact that not a single putrefactive bacteria showed itself in any of the cultures that were made. He examined the fluid with special reference to the presence of sup purative bacteria, particularly the staphylococcus pyogenus aureus, but none were found.

Here, then, was

AN ABSCESS WITHOUT SUPPURATIVE BACTERIA.

But the abscess as found was an old one, and probably when acute the suppurative bacteria were present and started it, and then died, the suppuration continuing. The suppuration continued because the cavity was lined throughout with granulation tissue that doubtless originated in an acute process. Cases of this kind would seem to be of practical interest and importance surgically, because of the possibility that such a purulent fluid as this might gain access to the peritoneal cavity without the establishment of a septic inflammation, as, judging from the absence of bacteria, there would be no probability of its doing in this case.

Dr. J. W. Roosevelt presented a specimen of

MALIGNANT STRICTURE OF THE ŒSOPHAGUS WITH CONTRACTION OF THE STOMACH,

removed from the body of a woman, forty-nine years of age. The previous history was that of chronic gastritis. She never vomited blood. About six months before death the pain from which she had suffered disappeared entirely. The constant vomiting of food was almost the only symptom. The small-sized stomach-tube could not be introduced. After that she was rather constipated, this peculiar vomiting continued, and she lost flesh ; but, on the whole, the patient did very well. Two weeks before death a small sound could not be passed through the stricture, yet the woman drank fluids and retained them, even as much as a quart at a time, but within half an hour or so the water would be regurgitated, practically unchanged. She lived on milk, however, taking solid food occasionally, which would return within two or three hours after it had been swallowed.

At the autopsy the œsophagus for nearly its entire length was found dilated to about the size of a quart champagne bottle. The stricture consisted of a ring of nodules surrounding the cardia, and the stomach was a little less than six inches in length and two inches in diameter. There were no secondary deposits.

TELANGIECTATIC SARCOMA IN THE MESENTERIC GLANDS—INTRACANALICULAR FIBROMA OF THE MAMMÆ—MYOMA OF THE STOMACH—FIBROMA OF THE UTERUS—FOLLICULAR CYST OF THE OVARY—CHRONIC INTERSTITIAL OÖPHORITIS—HEALED FRACTURE OF THE RIBS—PERFORATING ULCER OF CORNEA WITH PROLAPSE OF IRIS.

Dr. Ira J. Van Gieson presented specimens removed from an adult well-nourished dog.

The two lowermost mammary glands on the right side were replaced by dense, mottled, brownish-white masses, two by three centimetres in diameter. The remaining glands on the right side contained similar masses, but smaller. Contents of thorax normal. On the inner surface of the second, third, fourth, fifth, and sixth ribs there were a series of slightly projecting nodules at the junction of the anterior and the middle thirds. The intestines were normal. The mesenteric glands were swollen and soft. In the stomach there were two dense, white, distinct masses in the muscular coat elevating the mucosa. The liver was fatty in spots. The kidneys and the spleen were normal. In the uterus there was a mass of tissue, attached by a narrow pedicular base to the anterior wall, corresponding to the shape of the uterine cavity and nearly filling it. The left ovary was cystic ; the right ovary was shrunken and hard. On both sides the eyelids were glued together. There was pus between the lids and the cornea. There was a perforation of the cornea near the centre on both sides. The brain was normal.

Microscopical examination.—In the mesenteric glands there were a large number of blood-vessels, with thin, delicate walls, surrounded by rather closely packed clusters of small spheroidal cells. The cells nearest the walls of the blood-vessels were cuboidal. Sections of the mammary glands showed a growth of new connective tissue in the ducts, and extending into the acini as pediculated masses. The process was developed to a moderate degree only, and many acini, although slightly dilated, were free from such masses. Scattered through the section were collections of pigment of hematogenous origin. The masses in the stomach consisted of rather loosely packed, smooth muscle-fibres. The tissue in the uterus was composed of fibrous connective tissue covered with epithelium. The left ovary was changed to a fibrous shell enclosing a granular mass of detritus. The right ovary was reduced to a dense mass of connective tissue containing blood-vessels. The nodules on the ribs were caused by the non-apposition of the two ends of the ribs, which have been fractured at these points.

The President remarked that the specimens were especially interesting with reference to the existence of intracanalicular fibroma, which was of infrequent occurrence, and when present was very liable to be mistaken for carcinoma. The case was furthermore interesting on account of the large variety of tumors present.

Dr. Porter remarked that dogs were very liable to sarcoma.

The Society then went into executive session.

Stated Meeting, February 9, 1887.

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

DR. G. C. FREEBORN demonstrated under the microscope the results of

TWO NEW METHODS OF STAINING ELASTIC FIBRES,

namely, Lustgarten's and Herxheimer's. Lustgarten (*Fortsch. d. Med.*, iv., 1886, No. 22, p. 734) employs for this purpose Victoria blue, a basic aniline dye, from the Baden Soda and Aniline Manufacturing Company.

Sections of tissue hardened in alcohol do not give satisfactory results, but fresh tissue hardened in Flemming's solution for twenty-four hours and then further hardened in alcohol gives sharp pictures.

Sections of tissue, hardened as above, are placed in a mixture of one part of a concentrated alcoholic solution of Victoria blue and four parts of water for twenty-four hours. The sections are then removed from the staining fluid, washed quickly in alcohol (five to ten seconds), drained of the superfluous fluid by placing them on a bit of filter paper for a few seconds, cleared in oil of bergamot, and mounted in Canada balsam.

Connective tissue and cells stain a faint green, nuclei a darker green, elastic fibres a light green, but darker than the other elements. In the specimens under the microscopes he had not succeeded in obtaining the exact shades described by the author. In his specimens the tint was bluish. This difference in color might be due to the grade of the dye employed, as he had found that the Baden Company manufacture three different shades. The dye used by him is known as the medium shade.

Herxheimer (*Fortsch. d. Med.*, iv., 1886, No. 24, p. 785) hardens fresh tissue in Müller's fluid, though good pictures may be obtained with tissues hardened in alcohol, picric acid, or Flemming's solution.

As a staining solution he uses the following mixture: 1 c.c. of hæmatoxylin (best obtained from Grübler in Leipsic), 20 c.c. of absolute alcohol, 20 c.c. of water, 1 c.c. of a saturated solution of lithium carbonate in water. Alum solutions of hæmatoxylin, Heidenhain's solution, or Weigert's solution may be used. The specimens shown were stained with Weigert's solution.

The sections, which should not be over 2 to 100 mm. thick, are placed in the staining solution for from three to five minutes. They are then removed and placed directly in a dish of the officinal solution of the chloride of iron for from five to twenty seconds. In this solution an iron lake is formed with the hæmatoxylin, and immediately afterward the decolorization takes place. Too prolonged action of the iron solution will remove the color from the finest fibres. After removing the sections from the iron solution, they are well washed in a large dish of water, when clouds of color will be given off from the sections. Then pass through alcohol twice, clear in oil of cloves, and mount in Canada balsam. The iron solution and the alcohol should be frequently renewed.

The elastic fibres stain a blue-black to a deep black color, and stand out sharp against the underlying connective tissue, which is stained more or less of a light gray, often running off into a bluish tint. Nuclei sometimes stain of a grayish tint.

VERTEBRAL CARIES—LAST CERVICAL AND FIRST SIX DORSAL—PARAPLEGIA—RECOVERY OF PARAPLEGIA—ENLARGEMENT OF RIGHT HEART—HEMORRHAGIC INFARCTION OF LUNGS—DEATH.

DR. JOHN F. RIDLON presented the spinal cord of a patient who died in St. Luke's Hospital on the 7th inst.

The history was as follows: Mary D——, six years of age, was admitted October 24, 1883, to the orthopædic service under the care of Dr. Newton M. Shaffer and himself. Both parents died of consumption. She had three healthy brothers, and two brothers died from causes unknown. She had four healthy sisters. Other hereditary history not known. The curve in upper dorsal spine was first noticed in January, 1881—six years ago. From that time the curve gradually increased in spite of treatment by a brace at one of the public institutions in this city for the treatment of cripples. On her admission to St. Luke's Hospital, three years and three months ago, her general condition was fairly good. There was a large kyphosis which involved the four first dorsal vertebræ. There was paraplegia to the following degree: She could not stand alone; there was very little voluntary motion in the lower extremities; and all the tendon reflexes of the lower extremities were greatly exaggerated. Sensation seemed normal. No bladder or rectal symptoms.

From that time, October 24, 1883, to June 29, 1885, there was no treatment except rest in bed. Meantime the paralysis of the lower extremities was complete—there was not the slightest control over a single muscle. From this time on, the same conservative treatment being pursued, there was a gradual and steady gain. On

March 10, 1886, she was able to move the toes somewhat, flex the feet to a degree, and flex the legs on the thighs. On April 6th she walked about forty feet. Reflexes were reduced almost to normal. On April 14th Taylor's antero-posterior spinal support and chin-piece with Shaffer's ball-pivot joint was applied and the patient was allowed up. On April 16th, after being up two days, she seemed so unsteady on her feet that she was again put in bed, the brace being kept on without the chin piece. On October 20th she was again allowed up for five minutes twice a day. December 1st she was able to walk alone the length of the ward and back. There was a slight exaggeration of the tendon-reflexes in the lower extremities. December 22d, allowed up half an hour twice daily. January 27, 1887, began to have difficulty in breathing. She was under the care of Dr. A. B. Ball till February 1st, and of Dr. F. P. Kinnicutt from that time till her death, at 3 A.M., February 8th.

Post-mortem examination by Dr. Frank Ferguson, February 8, 1887. Body emaciated. Rigor mortis slightly marked. Spinal curvature and projection forward of the breast. Diaphragm at the sixth rib on either side. Loose old adhesions generally distributed over both lungs. Tubercles at apex of left lung. Areas of hemorrhages of small size. Mucus and pus in the bronchial tubes. Old phthisis at apex of right lung. Numerous hemorrhages (infarctions) of considerable size in lower lobe of right lung. Heart was larger than normal. Left ventricle moderately dilated; its walls thin; its valves normal. Right ventricle enormously dilated; its walls thin; its valves normal; its muscular tissue showed fatty degeneration. Spleen normal in size, firm in consistency, dark in color; kidneys congested, firm in consistency, capsule not adherent, surface smooth, markings distinct. Both organs were alike. Mesenteric glands moderately increased in size. Stomach, pancreas, and gall-bladder normal. Liver: Peripheri of acini were anæmic and fatty. Urinary bladder normal. Caries of the spinal column involving the last—seventh—cervical and first five dorsal vertebræ. The angle of the curve was very sharp (so that the upper surface of the sixth dorsal and the under surface of the sixth cervical were not more than an inch and a half apart, and this space was filled in with a cheesy mass from the cord forward, there being but a thin shell of bone anteriorly). The cord was about half an inch in diameter, flat, and its gray substance was not distinguishable. Above and below the angle the spinal cord seemed normal.

[The specimen was referred to the Committee on Microscopy.]

Remarks.—Dr. Ridlon said that it was difficult to tell at the beginning whether recovery from the paraplegia attending Pott's disease would occur within two months or three years, and he knew of no method of treatment which would enable one to answer this question. Is there any treatment more satisfactory than rest in bed and the support given by a fairly good fixation apparatus? He had used iodide of potassium, and had become convinced that it produces no effect upon the paraplegia, unless there be reason to believe that hereditary syphilis is present.

To the naked eye the gray matter in the specimen presented was apparently destroyed, but whether or not the nerve-fibres had been destroyed he was unable to say.

He would also ask if anyone had seen a case in which paraplegia was present and the kyphosis was in any other region than the upper dorsal.

Again, were we to expect dilatation of the right side of the heart as the result, in these cases, which would give rise to a pulmonary condition that would terminate life?

Dr. Van Santvoord referred to a case which he treated with the actual cautery, while an interne in Bellevue Hospital. The cautery was applied to relieve pain, but the paraplegia began to disappear at once, and after a time the girl recovered from the paralysis.

Dr. Ira Van Gieson remarked concerning restoration of function after destruction of nerve-tracts that a recent observer had experimented under Stricker's direction by removing a portion of the motor tract in dogs, and had found that function was restored in from three to six weeks. The conclusion was that the restoration of function occurred through collateral channels in the motor tracts injured or destroyed.

Dr. H. J. Boldt presented a specimen of

PERSISTENT BRONCHO-PNEUMONIA

removed from the body of a child nine months old that died with symptoms of meningitis. There had been nothing to direct attention to the chest, and the child had been healthy until the development of the meningitis.

The lower portion of the left lung was adherent to the chest-wall and had one cavity filled with pus. All the other organs were healthy, except the brain, which showed simple meningitis.

METRITIS—PERIMETRITIS—ENLARGED GLANDS.

Dr. Boldt presented the pelvic organs removed from the body of a woman, forty-nine years of age, who came under his observation on October 1st, last, when she claimed that she had enjoyed good health until a few months previously. About one year ago a small tumor was detected in the right inguinal region, which was regarded as an ovarian cyst. When Dr. Boldt first saw her the tumor had reached considerable size, and he advised an operation, but it was declined, and the patient died of exhaustion.

At the autopsy the uterus was found enlarged, its walls thickened, and the entrances to the Fallopian tubes were obliterated. On the right side there was a cyst that had been bound down by adhesions, and upon the top of it the tube could be distinctly traced. On the left side it was impossible to determine, positively, by gross examination, the presence of either the tube or ovary. The retroperitoneal glands were enlarged, and it was thought that they might be sarcomatous.

The case illustrated the importance of early operation for removal of ovarian cyst.

The specimen was referred to the Committee on Microscopy.

Dr. W. H. Porter presented a pair of lungs which illustrated

ACUTE ANTHRACOTIC PNEUMONIA

in connection with acute lobar pneumonia. He had seen four or five such cases, all occurring in workmen on the new aqueduct. The symptoms had been chill followed by moderate rise of temperature and continuation of febrile movement for a few days, and finally the symptoms all abated and the patients were about the wards of the hospital in a very comfortable condition, but died suddenly on the development of dyspnœa and pulmonary œdema.

The anthracotic part was apparently an acute development of this peculiar lesion. He had seen two or three cases in which this anthracotic condition of the lungs only was found, without evidence of inflammatory exudate into the air vesicles. Whether or not the anthracosis predisposed to the occurrence of lobar pneumonia, he was unprepared to answer ; it might be that these persons had the lobar pneumonia because of the exposure incident to their work.

The President remarked that this was an interesting class of cases, especially in connection with the new notions of the pathology of acute lobar pneumonia. Although the particular bacterium which causes this form of pneumonia has not been established, and it is not improbable that there may be several capable of causing it, the general impression is that there are micro-organisms which bear the relation of cause and effect to this disease; but it also remains that there are predisposing factors which are equally important, and perhaps there is a peculiar exposure that predisposes to acute lobar pneumonia. Here, however, is another cause, apparently, and the acute irritation produced by the little foreign particles may be a predisposing factor. In that respect, especially, the class of cases mentioned by Dr. Porter becomes one of great interest.

Dr. Porter had thought that the acute lobar pneumonia in these cases was the result of the acute anthracosis, without reference to bacilli.

DILATED URETERS.

Dr. J. S. Thacher presented two specimens. The *first* was found at autopsy in the body of a woman. The cause of the dilatation was a collection of small polypoid outgrowths from the mucous membrane in the middle part of the ureter and over a space an inch or an inch and a half long. The dilatation of the pelvis of the kidney was not extreme. The little growths, which were numerous, were not fibroid in character, but rather cystic, containing granular matter and squamous epithelia.

The *second* specimen was from the body of a fœtus six months old, and the ureters were dilated to about the size of the thigh of the embryo at that period. The pelves of the kidneys were slightly dilated. The bladder was enlarged, its walls hypertrophied and quite firmly attached to the anterior wall of the abdomen. He was unable to find any cause for the retention of the urine.

Dr. Ira Van Gieson presented a specimen which showed:

A FOREIGN BODY IN THE EYE—PANOPHTHALMITIS FROM INJURY.

Three weeks before enucleation the patient was struck by a twig, penetrating the sclera about one centimetre posterior to the sclero-corneal junction and passing through the globe to the diagonally opposite ciliary body. The foreign body was thorn-shaped, rather sharply pointed, and measured one and a half centimetres in length.

Microscopical Examination.—The foreign body was surrounded by a zone of granulation tissue. The lens was absent and the collapsed capsule surrounding a small mass of new connective tissue occupied approximately its normal position. Just posterior to the foreign body was the detached retina. Between the detached retina and the optic nerve entrance was a mass of extravasated blood. The anterior chamber was filled with pus. The iris and ciliary body were infiltrated with pus-cells.

DR. R. VAN SANTVOORD presented a specimen of

POSSIBLE TUBERCULAR DISEASE OF THE PHARYNX

removed from the body of a child eighteen months and nineteen days old. Five months ago it was noticed to be suffering from irregular attacks of fever, with cough and râles in the chest, and that it was emaciating somewhat. There was some difficulty in swallowing at one time, but nothing special to explain it was found. The child died four days ago. At the autopsy there were found two ulcers, one in each pyramidal pouch, and about an inch in length. There were also ulcers on either side of the external surface of the epiglottis, about one fourth of an inch wide. On the posterior wall of the pharynx were irregular ulcers, and one small point of ulceration in the left tonsil. The process did not extend into the nose. The bronchial glands were enlarged, but were not cheesy. The upper and the lower lobes of the right lung were solidified largely by broncho-pneumonia, and scattered through the consolidation were yellowish spots, which which were apparantly tubercular nodules that were becoming cheesy. Nothing especially noticeable was found in the abdominal contents.

About fourteen months ago he presented a similar specimen, and in that case difficulty in swallowing was noticed only four days before the death of the child. The lesion had been described as occurring in adults, but whether it was frequent or not in children he was not prepared to say. The specimen was referred to the Committee on Microscopy.

DR. RIDLON had been led to believe, from clinical observation, that lesions of this character which it had been customary to regard as tubercular occurred without any hereditary history of tubercular disease, but where there was a positive history of syphilis.

DR. A. P. DUDLEY presented a specimen of

CANCER OF THE UTERUS

that developed upon the site of an old cervical laceration in a woman forty-eight years of age, who had borne eight children, and had been attended by a midwife. She was not aware that laceration had occurred. The malignant disease was quite far advanced, and was removed by the vaginal operation. Considerable difficulty was experienced in the operation because of the attachment to the uterus of two fibroids, and the mass was finally removed in sections by means of the elastic ligature.

The patient was under the influence of ether nearly three hours. She did fairly well until the following morning, when it was found that there was suppression of urine, and death occurred thirty-eight hours afterward.

Dr. W. H. Porter made the autopsy, and found that the entire peritoneal cavity was absolutely free from suppuration. The kidneys were in a condition of rapidly advancing acute parenchymatous metamorphosis, and the liver was in the same condition, and also cirrhotic. He had seen twelve or thirteen similar cases, in which this acute disease of the kidneys had developed after severe surgical operations, while everything about the site of the operation indicated a reparative process, but the symptoms were those ordinarily ascribed to septicæmia. In these cases the urine contained neither casts nor albumen before the operation, but that which was in the bladder at the time the autopsy was made contained both. He had therefore been led to believe that, in a large number of cases, what had been called septic symptoms had been due to renal changes. He regarded prolonged etherization as unsafe, and, if there was evidence of renal disease, as contraindicated.

Dr. Dudley also presented

A SLOUGHING FIBROID

removed from a patient, aged forty-three, who had borne ten children. She had suffered from pain and hemorrhage for nearly a year before he saw her. There was double laceration of the cervix. The fibroid was removed by means of the spoon-saw from the anterior wall of the uterus, close to the fundus.

The patient had intermittent heart-beat, and also an aortic murmur, besides about ten per cent. of albumen in the urine, which cleared up under the influence of citrate of potash and infusion of digitalis.

This patient took a small quantity of ether without unfavorable symptoms, and he intended to again administer it and subject her to an operation for repair of her perineum.

UTERINE POLYPUS, WITH ENLARGEMENT OF THE ORGAN, TWO YEARS AFTER THE MENOPAUSE.

Dr. Dudley also presented a small polypus removed from the anterior wall of the body of the uterus of a woman, aged fifty-four, who had been past the menopause two years. The cervix was really a senile one, and he divided it sufficiently to enable him to dilate and remove the polypus that he had previously recognized lying within the cavity of the cervix. The uterus measured four inches in depth.

Dr. Boldt referred to three cases in which acute renal changes were found, while the symptoms were supposed to be due to septicæmia. He also recalled two cases of polypus occurring after the menopause.

The Society then went into executive session.

Stated Meeting, February 23, 1887.

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

THE REPORT FROM THE COMMITTEE ON MICROSCOPY, on the uterus and its appendages presented by Dr. H. J. Boldt at the last stated meeting, was submitted, as follows:

Sections were made from one of the retro-peritoneal glands. Its perivascular spaces were found to be irregularly dilated, and packed full of irregularly shaped epithelial cells. The epithelial cells within the alveolar spaces were not separated by any intercellular substance, and they had no definite arrangement. Between the masses of cells the fibrillated connective tissue was increased in thickness, and infiltrated with new cells. Carcinoma.

G. C. FREEBORN,
WILLIAM H. PORTER.

ACUTE ANTHRACOTIC PNEUMONIA.

DR. WILLIAM H. PORTER presented sections made from the lungs removed from a case of acute anthraccotic pneumonia, with the following report:

Upon microscopical examination, the interlobular connective tissue was found to be very much thickened and in a state of œdematous infiltration. This thickened tissue was also infiltrated with inflammatory cells, also small and large round connective-tissue corpuscles. That this very apparent interstitial thickening was chiefly œdematous in character was proved by its rapid decrease in passing the sections through the absolute alcohol.

The air-vesicles were filled with the ordinary inflammatory exudation common to lobar pneumonia, the larger portion of which was in the stage of gray hepatization, but a few of the air-vesicles still contained a delicate reticulated net-work of fibrillated fibrin.

The most interesting point was the very large amount of black and apparently inorganic pigment which was found in large quantities in the inflammatory exudation into the air-cells, while the interstitial tissue contained comparatively little.

No pneumococci could be found. In these cases both the interstitial and intravesicular inflammation apparently was due to the large amount of inorganic pigment drawn into the lungs. In these particular instances the evidence is strongly against an infection or germ as the cause of the pneumonic process, and most decidedly favors a direct injury as the causative agent.

PISTOL-SHOT WOUND OF THE BRAIN.

Dr. Porter presented a second specimen, with the following history: Male, twenty-three years of age, German, who had been to a ball the night before. He returned early in the morning, and shot himself without apparent cause. Death followed in about two hours from hemorrhage.

At the necropsy the track of the ball was found to be from right to left completely through the skull. The direction was such that both right and left middle meningeal arteries were divided. The bullet-holes in the bony walls were about three-eighths of an inch in diameter, while that through the brain-substance was about one inch in its diameter. The course of the ball through the brain was from right to left, just in front of and below the lateral ventricles. There was a stellate fracture at the vertix, and both orbital plates were fractured; also the occipital bone on both sides of the foramen magnum. The special points of interest were the complete division of the middle meningeal artery upon both sides; the small bullet-hole in the skull, and its large diameter through the brain substance; the slight amount of intracranial hemorrhage, and the large number of cranial fractures.

TRICUSPID AND BICUSPID INSUFFICIENCY—AORTIC STENOSIS — CARDIAC DEFORMITY — INTERSTITIAL PNEUMONIA—AND INTRALOBULAR CIRRHOSIS OF THE LIVER.

Dr. Porter presented a third specimen, with the following history by Dr. William A. Moore, house physician:

C. R. H——, twenty-four years of age, was first admitted to the service of Dr. A. H. Smith, at the Presbyterian Hospital, January 8, 1887, and later to the service of Dr. William G. Thompson. He gave a family history of both phthisis and cardiac disease. He has had repeated and severe attacks of rheumatism; the last, which was unusually severe, occurred five years ago. He has also had pleurisy and typhoid fever. For a number of years he has suffered with dyspnœa and cardiac trouble. The present sickness commenced December 19, 1886, as a severe cold. When admitted he was very anæmic and considerably emaciated. His feet and legs were œdematous.

Physical examination revealed a dilated, a rapid and tumultuous heart's action, and a widely diffused apex-beat. Upon auscultation a distinct bicuspid and tricuspid regurgitant murmur was heard; also a slight aortic obstructive murmur. There was marked jugular pulsation.

The patient gradually sank, and died from exhaustion and cardiac failure.

Necropsy twenty-four hours after death. Body very much emaciated; rigor mortis marked.

Thoracic cavity: The pericardial sac was completely obliterated by an old and fibrous pericarditis.

The left auricle was dilated and its walls very much thickened. The endocardium looked like dense white, fibrillated connective tissue. One segment of the mitral valve was shortened and bound down to the ventricular wall. The opposing segment was elongated and thickened.

The left ventricle was about normal in size, but its muscular wall was thin and degenerated. The segments of the aortic valve were the seat of numerous small vegetations. The aorta was the seat of extensive endarteritis and fatty infiltration.

The cavity of the right ventricle appeared to be very much diminished in size; the segments of the tricuspid valve shortened and adherent to the cardiac wall, thus nearly obliterating their usefulness. The right auricle was very much dilated, its endocardium in a state of fibroid thickening similar to that of the left auricle. The muscular coat was greatly thickened and reticulated. Both lungs were firmly bound to the chest-wall by old pleuritic adhesions. There was a marked interstitial thickening, some congestion and œdema of the pulmonary substance, also a few zones of broncho-pneumonia.

Abdominal cavity: The *spleen* was normal in size. Its capsule was thickened by fibrous patches. The *kidneys* were the seat of a moderate amount of chronic diffuse nephritis. Their pelves were the seat of punctate hemorrhages and were deeply congested. Weight of each six ounces.

The *liver* was enlarged, weighing sixty ounces. Its external surface was covered with patches of fibrillated connective tissues, and fringes of the same hung from its external surface in many places. There was a marked intralobular cirrhosis, and numerous microscopic gummy formations in the intralobular spaces.

The chief points of interest are those in connection with the heart, viz., extensive lesions upon the right side are very uncommon. Out of two thousand necropsies only two have given evidence of primary changes on the right side. As a late secondary lesion to organic valvular changes on the left side, a degeneration and dilatation of the right ventricle causes both pulmonary and tricuspid insufficiency. But organic changes causing valvular lesions of the right side are extremely rare. From the almost universal absence of any organic valvular change in the pulmonary and tricuspid valves, and from the well-known physiological fact that the tricuspid valves are always insufficient, we can, practically speaking, exclude valvular lesions of the right side of the heart when making a physical examination of the heart.

MULTIPLE AND RETAINED GALL-STONES—PARENCHYMATOUS METAMORPHOSIS OF THE LIVER AND KIDNEYS—MARKED JAUNDICE.

DR. PORTER presented a fourth specimen, with the following history abstracted from the very complete one taken by Dr. William A. Moore, house physician: Mrs. M. F——, aged thirty two, Irish, laundress, was admitted to the service of Dr. William G. Thompson at the Presbyterian Hospital. There was a clear history of repeated attacks of biliary colic and severe facial neuralgias, otherwise her previous history was good. Her present attack commenced on February 1, 1887, with a severe pain in the right side, and the next day she commenced to menstruate. The pain continued, but was intermittent in intensity. Two days before entering the hospital she had a severe chill, followed by a high fever.

February 12th she was admitted, and was found upon examination to be a large and very fleshy woman, the skin had an ashy hue, and she had every appearance of being a very sick person. The conjunctivæ slightly jaundiced; none of integument. Physical examination negative.

She remained in the hospital until February 21st, with symptoms which pointed toward occlusion of the common bile-duct, such as biliary colic, progressive jaundice, clay-colored stools, and high-colored urine. There were frequently repeated chills. The temperature ranged between 100° F. and 107° F., but most of the time it was above 103° F. It rose after each chill, but would fall for a time after thirty grains of antipyrin were taken.

General sponging of the body kept the temperature more evenly down.

On the 17th exploratory aspiration of the hepatic region, but no pus was obtained.

On the 19th albumen appeared in the urine, followed the next day by casts. The amount of urine voided was fair in quantity. Stimulants were freely administered, but all the symptoms grew worse. Her mental faculties remained clear to last, but heart gradually failed, and death appeared to be due to uræmic poisoning, œdema of the lungs, and cardiac paralysis.

Necropsy twelve hours after death. Rigor mortis marked. Body deeply stained yellow, as were all the tissues. Adipose tissue abundant. Thoracic cavity: The pericardial contained one-half ounce of yellow, serous fluid. The heart was soft, flabby, and its cavities dilated; weight twelve ounces; valves normal. Microscopically the muscular fibres were found to be in an advancing state of granular metamorphosis. The left lung was slightly adherent to the chest-wall, but there was no fluid in its pleural cavity; weight eighteen ounces. The right lung was slightly bound to the chest-wall at its apex by old adhesions. At the ninth intercostal space, about midway between the axillary and anterior scapular line, there was a marked patch of punctate subserous ecchymosis, with a slight amount of blood-stained fibro-plastic exudation. The same condition was noticed in the pulmonary and diaphragmatic pleura. There were a few ounces of blood-tinged serum in the right pleural cavity; weight of right lung eighteen ounces. Both lungs were congested and œdematous. Abdominal cavity: The peritoneal cavity contained about one pint of fluid blood and about the same amount of blood-clot. This firm clot lay between the liver and the posterior abdominal wall. There was also considerable retro-peritoneal hemorrhage. There was no peritoneal inflammation. The spleen was large and soft; weight ten ounces. Kidneys: Both glands were very much enlarged and softened; their capsules non-adherent. The cortex was thickened, the markings straight. Upon microscopic examination the epithelium was found to be in an advancing stage of granular metamorphosis. There was no evidence of an inflammatory action, but there was considerable bile-pigmentation. There was a granular condition of the os uteri, also an inflammation of cervix and fundus uteri. There was cystic degeneration of both ovaries, also a pedunculated fibroid of the fundus, one inch in diameter; alimentary tract normal. The duodenum was opened in situ, and the common bile-duct found to be patent, but just within its orifice it was widely dilated. All the hepatic tributaries were dilated, and contained small gall-stones. The gall-bladder was thickened and punctated. The gall-bladder and the hepatic tributaries contained, in all eighty-six gall-stones, ranging in size from one eighth of an inch to one inch or more in diameter. The liver showed marked parenchymatous change and granular metamorphosis, but no positive inflammatory or suppurating foci.

EXSECTION OF THE HEAD OF THE FEMUR.

Dr. L. H. Sayre presented the head of the femur and a portion of the acetabulum removed from a boy thirteen years of age. The operation was performed February 21, 1887. The family history of the patient was good, and he had always enjoyed a fair degree of health. It had been noticed, however, that all through his life he had walked with a peculiar movement about the hip, and, as described by his sister, "as if he had a wooden leg."

Three years ago he had a severe fall, striking upon his right hip, and two years ago his lameness had become so marked that he was sent to a surgeon on account of supposed morbus coxarius. After examination it was concluded by the surgeon that the case was one of reflex trouble, and the patient was circumcised. The improvement after the operation was very pronounced, and for eighteen months the boy walked about as he had always done before receiving the fall.

On last Thanksgiving Day, after exposure, he complained of slight pain in the hip, became somewhat languid, and at the end of six weeks a marked deformity was present, yet it differed from that seen in the third stage of hip-joint disease. The leg was slightly flexed upon the thigh and the limb was adducted across the middle third of the opposite thigh, but the foot was not rotated inward. There was slight tenderness on pressing the head of the femur into the acetabulum, but no tenderness on pressing the bone upward. There was slight motion at the joint, but it was apparently nearly anchylosed. The entire buttock on the right side was enlarged and indurated. At no time had the boy suffered from the pain usually associated with hip disease.

The diagnosis was probable

OSTITIS OF THE ILIUM, INVOLVING THE ACETABULUM ;

the condition of the head of the femur was not made out satisfactorily. The patient was put to bed and made more comfortable by the adjustment of an extension apparatus.

About two weeks ago Dr. Sayre detected deep-seated fluctuation over the gluteal region. The patient had then begun to lose his appetite and to have irregular chills. Dr. Sayre then made an exploratory incision, and found an abscess from which he evacuated four or five ounces of pus, which was without débris and did not present the appearance usually shown by pus associated with dead bone. The capsule of the joint was ruptured, and, under anæsthesia, the movements of the joint were quite free.

At this stage of the operation it became a grave question whether to simply drain the abscess or to explore concerning the condition of head of the femur and, if necessary, perform exsection.

On manipulation, however, a small quantity of the peculiar gelatiniform material seen with chronic bone-disease oozed from the ruptured capsule, and it was at once decided to exsect.

After the upper part of the femur was removed, it was found that the whole of the articular cartilage had been loosened from the head of the bone; and that the carious process had extended through the head of the femur entirely. The acetabulum was carious at the upper portion, and from about the insertion of the ligamentum teres he removed dead bone down to the internal periosteum, but did not enter the pelvis. The cavity of the abscess was cleaned out thoroughly, a drainage-tube was introduced, and the wound was closed. Since the operation the patient had been doing well; pulse, 100; and temperature, 99° F.

Dr. J. F. Ridlon cited a case which he saw first about five years ago, and in which recovery had taken place. He had been in the habit of treating these cases by other means than exsection, with the expectation of getting good results, hence he was specially interested in seeing the head of the femur removed at this stage of the disease. The symptoms, in his case, began with a little limp, following hemiplegic chorea, that continued without pain, tenderness, or reflex muscular spasm for nearly a year.

The case was watched very carefully by a competent orthopædic surgeon, and finally a traction-apparatus was applied. About one year and a half after the first symptoms were noticed, a small abscess was found in the gluteal region just below the great trochanter and behind, which gradually increased in size until it was as large as half a medium-sized orange, and then disappeared.

He mentioned the case for the sake of asking Dr. Sayre if he had seen abscess in the gluteal region except where there was perforation, as in his case, through the head of the bone rather than the capsule of the joint. His own belief was, and always had been, from clinical observation, that the abscess of hip-joint disease usually opened in front when it opened through the capsule, but when it opened through the bone the opening was outside of the capsular ligament, as in this case.

He would also inquire how long the abscess had existed when it opened. Four or five days ago he saw a case of hip-joint disease in which the abscess showed itself first in the anterior part of the thigh, one year ago last November. No apparatus was applied; the case was, substantially, let alone, and the abscess gradually increased in size until last Saturday, when it ruptured. At that time it occupied the upper third of the anterior portion of the thigh and the posterior part of the thigh to the extent of about half-way down to the gluteal region. It discharged only a very small quantity of pus, and on the following Monday fluctuation could not be made out. There had been no pain for a year and two or three months.

He regarded the absence of pain as of no importance at all in the early diagnosis of disease of the hip-joint. He believed that pain was one of the later symptoms of morbus coxarius.

Dr. Sayre said that the case had been under his observation about seven weeks. When he first saw it the entire gluteal region was enlarged and indurated, and fluctuation was detected about three weeks ago. He had seen cases of hip disease without pain, and he had been of the opinion that the disease began in the bone, rather than any of the other structures of the joint, and gave rise to extensive changes in the osseous tissue. He had seen a case like that mentioned by Dr. Ridlon, in which the abscess was apparently absorbed, and the reason why he operated in the present case was because the boy had marked constitutional symptoms. He had never exsected a joint in the condition shown by the specimen, and should look with great interest for the result of an early operation. The cases in which he had seen exsection had been those which were of many years' standing. According to his experience the abscess in hip disease almost always opened upon the thigh, either anteriorly or posteriorly.

ABSCESS OF THE LUNG SIMULATING EMPYEMA, IN A CHILD THREE MONTHS OLD.

Dr. L. Emmett Holt presented the lungs of a female child, in which the whole right lower lobe was converted into an abscess-sac with a thin wall.

The history was as follows: The child, an inmate of the New York Infant Asylum, was first seen on October 23, 1886, it having been ill three days with pulmonary symptoms. The temperature had varied between 102° and 103.5° F. There was cough and accelerated respiration. Physical examination showed exaggerated resonance and respiration over the left lung, with a few coarse râles. On the right side the râles were finer, the breathing in places broncho-vesicular, and over the whole lung behind there was a shade of dulness. It was noted as being questionable.

On the eighth day the temperature, which had remained uniformly at the height mentioned (102° to 103° F.), fell to 100° F., and for the week succeeding scarcely rose above 101° F. The examination of the lungs on the eighth day showed on the left side only exaggerated resonance and respiration. On the right side there was moderate dulness, back and front, with diminished respiratory murmur over the lower lobe, and bronchial breathing at the apex.

He did not see the child again until the seventeenth day, when the house physician called his attention again to the case, as the "broncho pneumonia" (this had been the diagnosis up to this time) was not clearing up well, and the temperature had risen to 102° F. again. The general condition was excellent, however; the child was plump and well nourished, nursed well, had only a moderate cough, slight looseness of the bowels, but no night-sweats. One would not have suspected from the facies and general appearance that anything ailed the child.

The physical signs found now were, flatness over the lower three-fourths of the right lung, front and back; this line of flatness varied also with position; there was absence of respiration over this part, and bronchial breathing at the apex. The apex-beat could not be made out to be displaced. An aspirator was introduced near the inferior angle of the right scapula, and, much to the surprise of all present, the effusion was found to be purulent. Nearly three ounces of healthy pus was evacuated; it was odorless.

Four days later, on the twenty-first day, the signs pointed to refilling of the chest, and the aspirator was again introduced, but only half an ounce of pus could be obtained.

The general symptoms were not specially changed during this interval; the temperature remained low until the twenty-fourth day, when it rose rapidly until it touched 105° F. Symptoms of great dyspnœa, cyanosis, and prostration developed, and the child died during the night. When these new symptoms developed, the house physician twice introduced the aspirator needle, but obtained no pus.

Autopsy.—Body well nourished; head not opened.

Thorax.—On removing the sternum, the left lung was found collapsed and compressed; the chest was nearly filled by the right lung, which extended two inches to the left of the sternum, overlapping and slightly displacing the heart. There were tolerably firm adhesions over the whole right lung, but no effusion whatever in the pleural cavity.

THE DIAPHRAGM WAS DISPLACED DOWNWARD.

On the lungs being removed, the right lower lobe was found greatly enlarged and had a fluctuating feel. On being incised, about three ounces of thick pus escaped. The lobe was hollowed out and consisted of little more than the wall of an abscess. Posteriorly the living tissue was thinnest, being scarcely two lines in thickness. At no point was it much over one-fourth of an inch thick, except about the root of the lung. The inner surface of the sac was rough and irregular, and seemed to have no well-defined pyogenic membrane. The bronchi were carefully followed out, but no communication existed between any of them and the abscess. The right upper lobe was partly compressed and partly consolidated, some small patches of pneumonia being seen in its lower border. The left lung could be easily inflated, except at one or two patches of presumably old collapse. The bronchial glands were slightly enlarged, but not cheesy. The capacity of the abscess-sac was found to be about four ounces. The other organs showed nothing of importance—the heart was healthy; the intestines showed evidence of slight catarrhal inflammation; kidneys normal in size, slight cloudy swelling; liver somewhat fatty, not enlarged; spleen normal. No evidence of a suppurative process was found anywhere. The bronchi leading to the lobes of the right lung showed evidence of a slight catarrhal inflammation only. No foreign body was found, nor anything else to act as a local cause of the abscess.

The case, so far as the speaker's observation and reading went, was a very unique one, and the diagnosis made of broncho pneumonia, followed by effusion into the pleura, seemed fully justified by the symptoms. It was difficult to see how the differential diagnosis between such an abscess and empyema could be made.

Dr. Frank Ferguson mentioned a case of abscess of the right lung, with a smooth limiting membrane, occurring in an adult. There was a granular condition of the pleural surface of the lung over the abscess, but not very extensive adhesion to the chest-wall, and the cavity had communicated with a bronchus which admitted a probe about one-tenth of an inch in diameter. He thought that it was primarily connected with this bronchus. The abscess was in the lower lobe, and in the upper lobe were numerous miliary tubercles.

Dr. Ferguson presented a specimen of

STRICTURE OF THE ŒSOPHAGUS,

occurring in a child two years and two months old, who was admitted into the New York Hospital on February 17, 1887. Her family history was good, and she was perfectly well up to three months prior to her admission, when she swallowed a small quantity of caustic potash.

Since then she had not been able to retain any food, but constantly vomited at every effort to eat or drink. Her appetite was very good; her bowels were constipated. On admission she was found extremly emaciated. Whatever was given her to eat or drink was retained only for a moment and then she passed her finger into her mouth and regurgitated what she had taken.

Auscultation revealed that the fluids and solids taken did not travel the entire length of the œsophagus.

The passage of a bougie was interrupted at about the middle of the tube. The child rejecting everything given her by the mouth, was put on nutritive and stimulating enemata, which she retained when given in small quantity.

During her residence in the hospital her temperature ranged below one hundred, and her condition was such that operation was not regarded as advisable. She died thirty-four hours after admission.

The *autopsy* revealed a stricture of the œsophagus, about its middle, two inches in length, and admitting a probe one-eighth of an inch in diameter. The œsophagus above the stricture was dilated; below it, normal. There was much thickening of the wall of the œsophagus in the neighborhood of the stricture.

The stomach was very anæmic, but otherwise normal. The other organs also were all anæmic, but otherwise normal. The body was greatly emaciated.

The Society then went into executive session.

Stated Meeting, March 9, 1887.

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

DR. W. H. PORTER read the

REPORT OF THE COMMITTEE ON MICROSCOPY,

signed by Drs. J. S. Thacher and G. C. Freeborn. In the specimens of ulcers of the pharynx and larynx presented by Dr. R. Van Santvoord, February 9, 1887, the committee were unable to find any evidence of tubercle.

Dr. Porter also presented a collection of

SMALL CALCAREOUS CONCRETIONS FROM THE SPLEEN.

DR. H. J. BOLDT presented a specimen of what he called

SYPHILITIC SALPINGITIS,

because there was no history of gonorrhœa; but there was a history of chancre, and the woman was suffering from well-developed secondary symptoms at the time of the operation, which was performed on account of the severe pain from which the patient suffered. The wound and the patient did well.

Dr. Boldt also presented a specimen of

PROLAPSED CYSTIC OVARY,

removed from a woman who had been under treatment about one and a half year without obtaining relief. The symptoms were intense backache, headache, dyspepsia, pain in the hypogastrium and inguinal region, severe dysmenorrhœa lasting throughout the entire period, and distressing cardiac palpitation. It had been two or three months since the operation was performed, and all the symptoms had disappeared except the palpitation, which had been very much modified.

DR. FRANK FERGUSON presented a specimen of

CARCINOMA OF THE BLADDER,

removed from the body of a man, sixty-one years of age, a native of Ireland, a widower, and a painter by occupation. He was admitted into the New York Hospital on January 29, 1887. Two years previously he first complained of stinging pain in his testicles and severe pain in the region of the bladder during micturition. These symptoms had continued, and he applied at the hospital for treatment. He had never had gonorrhœa.

On admission he was weak and anæmic. Within the last two years he had lost considerable flesh. Micturition was frequent and painful. No. 22 steel sound was tightly grasped in the prostatic urethra. Examination of patient by the rectum revealed the prostate enlarged and indurated. The urine was alkaline and had a specific gravity of 1.014. It contained a trace of albumin. Microscopical examination revealed quantities of mucus and pus. He always suffered constant, dull pain, but his pain was quite severe during micturition.

During his residence in the hospital he passed daily from thirty-seven to sixty ounces of urine, micturating from ten to fifteen times. During the last ten days of his life he vomited daily a greenish-yellow fluid. He gradually and constantly failed, and died on the afternoon of the 15th of the following February.

The autopsy revealed the bladder moderately distended with urine, pus, mucus, and grumous material. There was a tumor at the base of the bladder, involving the entrance of both ureters. The tumor, on opening the bladder, appeared slightly raised above the mucous membrane, was circular in outline, and measured one and one-quarter inch in diameter. The connective tissue between the rectum and the bladder, and also the seminal vesicles, were invaded by the tumor. Microscopically, it was composed of fibrous tissue and epithelial cells; the fibrous tissue arranged into alveoli which were closely packed by epithelial cells of small size. The ureters were greatly dilated; the left more than the right. The outlet of the ureters could hardly be traced through the tumor. The pelvis of the left kidney was very greatly dilated; nearly the entire organ was destroyed. The pelvis of the right kidney was also dilated. The cortex of each organ showed the lesions of chronic diffuse nephritis. The stomach was congested, and its mucous membrane was thickened. The other organs were normal.

FOREIGN BODY ENTERING THE CRANIAL CAVITY THROUGH THE ORBIT.

Dr. J. A. Wyeth presented a piece of a fence-rail that Dr. Cross, of Decatur, Ala., four years ago, removed from the cranial and orbital cavities of a negro who had been thrown from a railroad train while in motion and pitched headlong into a pile of rails. As he went, head on, a piece of a rail was plunged into the right eye in such a manner as to push the eye out of the orbital cavity almost completely, it being necessary to clip off only a few connecting portions of lacerated tissue to separate it entirely. While dressing the wound the doctor felt something within the orbit, which he removed and found it to be a piece of wood two and three-fourths inches long, three-fourths of an inch in its greatest diameter, and in its smallest diameter one-half of an inch. After the piece of wood was removed some brain-matter oozed through the orbital cavity. The man recovered without cerebral symptoms, and subsequently suffered no inconvenience except that attending the loss of an eye. This case was an additional illustration of how severely the brain may be injured without producing serious symptoms.

Dr. A. Palmer Dudley presented a

TUMOR REMOVED FROM THE RIGHT PAROTID REGION

of a woman twenty-eight years of age, married six years, and the mother of two children. This was the third operation, the first being performed four years ago. The patient came to him from Dr. N. P. Dandridge, of Cincinnati, with the following letter, dated February 25, 1887:

"Mrs. A—— first came under my care in September, 1885, for a swelling in the right parotid region. Some eighteen months before, a small tumor had been removed by Dr. Porter, of Boston. Dr. Porter, in answer to a letter, stated that the growth was a small circumscribed fibroid tumor, distinctly incapsulated, and enucleated without difficulty. No microscopic examination was made, but no suspicion was entertained that the growth was anything but a simple fibroma. When first seen I found a distinct swelling beneath the cicatrix of the former operation, to which it seemed adherent. This swelling was distinctly movable on the underlying tissues, and when seen a second time, some two weeks later, seemed to have somewhat increased in size, and I advised its immediate removal. This was accomplished on September 30, 1885. The cicatrix and adjacent skin were included in the superficial incision, and the surrounding fat in the deeper part of the wound was freely removed. No circumscribed tumor was found, and no suspicious tissue was left behind. The mass removed was most carefully examined microscopically, and nothing but a somewhat dense cicatrix and fatty tissue found. The wound healed readily. For some months there has been a steady increase in the size of the growth, and it is now considerably larger than when removed, in September, 1885. For some months I have only seen Mrs. A—— at long intervals, and have noticed each time a steady increase in the prominence of the swelling."

The growth seemed to be encysted, but the sac was so attenuated and spread out that Dr. Dudley was unable to say whether or not he removed all of it. The specimen was referred to the Committee on Microscopy.

Dr. Dudley also presented

A LARGE FIBROID, TOGETHER WITH THE UTERUS AND ITS APPENDAGES,

which he had removed the day before. The woman did not know that she had it until about ten days ago, when for swelling of the right leg she sought medical advice.

She was forty years of age, had never borne children, and she had never been troubled with her menstrual function. As soon as she learned of her condition she insisted that an operation be performed, which Dr. Dudley did, and found the ovaries and tubes and uterus so intimately connected with the growth that it was necessary to remove the whole. There were no adhesions. It was removed through an incision extending from the pubis to two inches above the umbilicus. The pedicle was transfixed and ligated in parts, cut short, touched with carbolic acid, and dropped back. The temperature of the patient was 100°, and her pulse 90. No morphine required to-day.

[At the stated meeting, March 23, 1887, Dr. Dudley reported that the patient was convalescent. On the sixth day he attempted to move the bowels, and did not succeed until several hours had been consumed in the use of ox-gall enemata, turpentine stupes, and a Seidlitz powder. Septic symptoms developed at once, and his patient nearly died; all of which, he believed, would have been avoided, had her bowels been moved earlier. He believed that he had lost two patients from septicæmia due to what he thought he had learned in the present case—namely, neglect to move the bowels sufficiently early. He also believed that the septic symptoms which frequently develop on the fifth or sixth day after laparotomy were frequently due to consequences attending failure to move the bowels.]

Dr. Ira J. Van Gieson presented

A MISPLACED KIDNEY,

removed from the body of a male thirty years of age.

The right kidney was found in the pelvic cavity; lying against the sacrum, behind the peritoneum, the superior apex is on a level with the pelvic brim, and its internal border follows the median line of the bodies of the sacral vertebræ. The renal artery is given off from the aorta at its bifurcation with the hilus, is situated on the anterior surface, and runs from the superior apex to about the middle of the anterior surface of the hilus. The ureter is situated inferiorly, the artery superiorly, and the vein lying between. The ureter measures seventeen centimetres.

Dr. Van Gieson also presented a specimen of

ATROPHIC KIDNEY WITH DILATED PELVIS.

There was nothing in the history referring to the kidneys.

Autopsy.—Heart: slightly enlarged; valves normal; atheroma of aorta. Lungs: emphysema; œdema; chronic bronchitis. Liver and spleen normal. Left kidney surrounded by dense, adherent tunica adiposa, containing calcific deposits; the kidney is small, measuring six by three centimetres; the pelvis is dilated, containing an amber-colored fluid; the cortex and capsule measure but two to three millimetres in thickness; ureter normal. Right kidney slightly enlarged, capsule adherent; the cortex is congested and rough.

Microscopical examination.—The cortex in places is one to two millimetres in thickness; the capsule is thickened. In the cortex there is an increased amount of connective tissue. This cortex is composed largely of altered glomeruli. The capsules of the glomeruli are thickened, and the tuft of vessels is changed to a mass of connective tissue in places calcified. Many of the tubules are present containing casts; there is obliterating endarteritis. The right kidney shows a moderate degree of chronic diffuse nephritis.

DR. WILLIAM P. NORTHRUP presented a series of specimens removed from the body of a child that died of

GENERAL TUBERCULOSIS,

which were interesting on account of the extent and severity of the lesions.

Male mulatto child, aged two, returned to the New York Foundling Asylum from its hired nurse, in the city. It suffered from almost uninterrupted convulsions at this time; no diarrhœa nor vomiting. Two days later the child died. No further history could be gained, and no further symptoms were made out. On autopsy these extensive and severe lesions of general tuberculosis were found:

Brain.—The pia mater at the base was studded with fine, discrete, miliary tubercles. In diameter they were no more than the diameter of the shaft of an ordinary pin, and were difficult to see except where the meninges stretch across spaces, as at the optic chiasm and elsewhere. Along the vessels of the great fissures they were a trifle larger, thickly studding the membrane and gluing the folds to each other. In good light the ependyma showed the same-sized tubercles, still more difficult to see on account of the color of the surrounding tissues; ventricles not dilated. What was of most interest was a tubercular nodule in the right *flocculus of the cerebellum.* It was in size about a cubic centimetre. He regarded this condition as rare. In his somewhat extended experience in autopsies on tuberculous subjects, such tubercular deposits in the brain-substance had occurred but once or twice before, and never of the size of the specimen shown.

Lungs.—Both adherent throughout. Tubercular pleurisy on the left side. Bronchial glands extremely enlarged, cheesy, many of them fluid in centres, and a few wholly softened and fluctuating in their capsules. The lungs had a flabby, muscle-like feel, showing no tubercles on surface, but torn adhesions. On section, large, cheesy, rosette-shaped nodules were seen, varying in size from thirty to forty millimetres in diameter. These in appearance were much like the large tubercles seen on the costal pleura. Besides the discrete tubercles, there was also extended sclerosis and peribronchitis, probably made up largely or wholly of diffuse tubercle-tissue. Dilated bronchi, chronic and recent pneumonia, and œdema completed the picture of the lungs.

The *intestines* presented an equally serious lesion. The mesenteric glands were enlarged to the size of an ordinary native hickory-nut, and grouped closely into a nodulated mass. These were cheesy and partly diffluent. The agminate glands of the ilium showed ulcers occupying the entire site of the original gland, having deeply overhanging edges. In most cases the glands were wholly destroyed; in a few the swollen, cheesy, and softened glands still remained.

The *liver*, still more unusually, showed a few doubtful tubercles on the surface. The organ was enlarged and fatty.

The *spleen* was almost normal. Normal in size or a little undersized. No tubercles to be seen on the surface; two to four doubtful millet-seed-sized tubercles found.

The *kidneys* showed two to five similar undoubted tubercles.

The striking peculiarities of the case were (1) the large tubercular nodule in the cerebellum; (2) the extreme size of the bronchial glands; (3) the extent of the lesion, and the size of the mesenteric glands; (4) the somewhat infrequent occurrence of ulcers of Peyer's glands in tuberculous children, and in this case the great extent and advanced state of the ulcers; (5) the absence of tubercle in the spleen—this organ is usually involved greatly in general tuberculosis; and (6) the fact that there was tubercular meningitis with such extensive tuberculosis of the thoracic and abdominal organs.

It was more common to find the meninges intact where there was well-distributed early tuberculosis of the other organs. It was the common experience in children, as in adults, to find children of the negro race more subject to tuberculosis.

Microscopic examination showed tubercular tissue in the nodule of the cerebellum, and also in the intestines.

DR. J. W. ROOSEVELT showed some microscopic sections which illustrated the effect produced by

A NEW STAINING-FLUID,

which he thought was especially adapted to micro-photographic work. It does not undergo any change after once combining with albuminous tissue. As at present used it consists of about twenty-five drops of a saturated solution of ferrous sulphate to an ounce of water, and fifteen to twenty drops of pyrogallic acid.

The Society then went into executive session.

Stated Meeting, March 23, 1887.

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

DR. G. C. FREEBORN, from the Committee on Microscopy, reported on

THE SPINAL CORD

presented by Dr. J. F. Ridlon, February 9, 1887. The portion of the cord included between the second and seventh dorsal segments, measuring about seven centimetres, is transversely flattened, and has an increased consistency. In this portion of the cord there is destruction of both the gray and white matter. The walls of the blood-vessels are thickened, and the anterior portion of the dura mater thickened, and it also contains calcareous deposits. The cord, above and below the compressed portion, shows the usual lesions of secondary degeneration.

THE PRESIDENT presented a set of specimens accompanied by the following history:

EXTREME CIRRHOSIS OF LIVER, WITH CHRONIC PHTHISIS, CHRONIC DIFFUSE NEPHRITIS, AND TUBERCULAR PERITONITIS.

He was indebted for the epitomized clinical history of the case, and the report of the autopsy, to Dr. Ely, house-physician, First Medical Division, in Bellevue Hospital.

A married Irishwoman, forty-two years of age, came into hospital, January 27, 1887, with a history of morning sickness, anorexia, and nausea for five months. About Christmas there was abdominal distention, and she was tapped. After this the feet swelled and she had a slight, dry cough. On account of procedentia of long standing she has had painful micturition. Of late urine has been scanty. She has had spots before the eyes. She had no rheumatism; a good family but a marked alcoholic history. She was on admission anæmic, had œdema of the feet; the abdomen was distended with fluid, and the veins were enlarged. There was at both apices, behind, slight dulness, prolonged and high-pitched expiration, with exaggerated voice. The liver was small, extending from fourth rib to about one and a half inch from free border of ribs. Urine was 1.008; trace of albumin; fifteen ounces in twenty-four hours. She was tapped again on February 8th, and twenty pints of clear serum withdrawn, the catheter being left in for continuous drainage until the 13th. There was no more marked accumulation of fluid, but the patient became delirious, gradually failed, and died of exhaustion on March 21st.

At the autopsy twenty ounces of clear serum was found in the abdominal cavity. There were omental adhesions, miliary tubercles, and a little fibrin over peritoneal surfaces. There were tubercular ulcers of the small intestine. Kidneys: Capsule somewhat adherent; surface a little rough; cortex uneven, with obscure markings and yellowish streaks and patches (fatty degeneration). Spleen large and soft. Liver very small, weighing one kilogramme. Its surface is finely nodular, the small, projecting nodules being very yellow from fatty degeneration. The cut surface is rough, with broad and narrow bands of translucent connective tissue running between the islets of fatty parenchyma. There were partial adhesions of the lungs, which presented the lesions of general chronic miliary tuberculosis without cavities.

The anatomical diagnosis as already given was confirmed by the microscopical examination.

THE PRESIDENT also presented a specimen of

SMALL MULTIPLE ADENOMATA OF THE KIDNEYS.

A middle-aged man, without previous history, was run over by the horse-cars, receiving a severe lacerated wound of the leg, from which he died on the fourth day. The only noteworthy visceral lesions were those of the kidneys. These organs were small, capsules adherent, surface rough, cortex thin and rough, markings irregular. In the cortex of the right kidney was a white, dense mass about one centimetre in diameter at the broader side, wedge-shaped, and reaching from the capsule toward the medulla. The surface of the kidney at this place was slightly depressed. Projecting from different parts of the surface of the left kidney were five globular bodies —some white, others reddish-brown in color, and moderately firm in consistency, but less hard than the surrounding kidney tissue. These tumors were from seven millimetres to one centimetre in diameter, and were partly embedded in the cortical tissue. Although projecting from the surface of the kidney, the capsule extended over them. Several other small white and brown masses, looking somewhat like small, old, subcapsular infarctions, were scattered through the cortex of the left kidney.

Microscopically, all of the little tumors present alike the structure of adenomata. They consist of a moderately vascular connective tissue, stroma enclosing larger and smaller spaces. These spaces are mostly elongated, forming irregular tubules, which are either lined so as to form a lumen or completely filled with large cuboidal or polyhedral cells. The spaces in some parts of the tumors are larger and more irregular in shape, and are more or less completely filled with polypoid projections from the walls, covered with epithelium. The epithelium is in part finely granular, in part filled with small fat-droplets. The brown tumors owe their color to the deposition of blood-pigment in the epithelial cells, in the stroma, and in the kidney-tissue adjacent to them, and to undecomposed red blood-cells in the alveoli and tubules. While the larger tumors are sharply outlined against the adjacent kidney-tissue by a zone of squeezed and fibrous kidney-tissue, the smaller and apparently younger forms merge almost imperceptibly into the adjacent kidney-structure, so that intermediary forms between kidney and tumor tubules are readily made out. For the rest, the kidneys show the usual lesions of atrophied chronic diffuse nephritis.

Adenoma of the kidney is not of such infrequent occurrence as to be especially noteworthy; but this case is of unusual interest—first, because of the multiplicity of the tumors and their presence in both organs; second, because, microscopically, they show both of the structural forms which adenomata of the kidney may present, namely, the alveolar and papillary; and third, because of their uniformly small size.

PLEURO-PNEUMONIA IN AN INFANT.

Dr. J. Lewis Smith presented the thoracic viscera taken from the body of an infant that died at the age of eight months. Its parents were healthy. It did not present any marked degree of emaciation. About five weeks before its death an abscess formed in front of the left ear, which was opened, and discharged a couple of drachms of pus. The discharge from the abscess continued for nearly three weeks, when it gradually abated and soon ceased.

Two weeks before death the child became suddenly very feverish and restless, and vomited several times. Vomiting was not a prominent symptom after the first day and a half. From the sudden occurrence of the sickness, the temperature—104½° F. on the first day—and the rolling of the head, with more or less of twitching movements, the attending physician supposed that the case was one of *cerebral disease*. There was slight cough, and Dr. Smith discovered slight dulness over the apex of the right lung.

He did not see the child again until about a week afterward, when he was surprised to find complete dulness over the entire left lung, such as accompanies pleuritic exudation. The respiratory sounds were indistinct and distant, and faintly broncho-vesicular. The pulse was 180, respiration 76 to 80, and the apex-beat could not be detected. The temperature throughout was elevated; at one time 105½° F., and scarcely below 103° F. On the day of Dr. Smith's second visit the temperature had fallen to 101½° F. He was also inclined to the opinion that there was

PERICARDITIS WITH THE PLEURISY.

The autopsy revealed a nodule of pneumonic consolidation in the upper part of the right lung, with a small amount of fibrinous exudation along the free border of the upper lobe. On cutting into the lung immediately under the fibrinous exudation there were found seven or eight little spots of apparently necrotic tissue. When the left pleural cavity was opened, about two and a half ounces of sero fibrinous fluid escaped, and the entire lung was covered with a plastic exudation. The lower lobe of this lung was almost completely hepatized by pneumonia. The superficial portion, besides, appeared to be in the condition of necrosis. The bronchi, the trachea, and the larynx showed only a very slight redness of the mucous membrane. Dr. Biggs had examined the specimens, and expressed the opinion that the pulmonary consolidation was probably of septic origin and secondary to the abscess. The heart was normal. The other organs were not examined.

The specimen was referred to the Committee on Microscopy.

DR. H. J. BOLDT said that he had made a sufficient number of post-mortem examinations in children who had exhibited marked cerebral symptoms with pneumonia to convince him that cerebral disturbance was of very frequent occurrence, and was of no special pathological importance.

DR. SMITH said that marked cerebral symptoms frequently occurred with the development of

PNEUMONIA AT THE APEX OF THE LUNGS IN CHILDREN.

DR. W. M. CARPENTER believed that marked cerebral symptoms were so frequently the precursor of pneumonia in children that a failure to examine the lungs was inexcusable.

DR. MARY A. DIXON JONES presented a series of ovaries, the seat of

CHRONIC OVARITIS, WITH CIRRHOSIS AND MORE OR LESS DISEASE OF THE FALLOPIAN TUBES.

The histories of five cases were read in which she performed laparotomy for the removal of these organs. All the patients recovered, and were apparently benefited by the operation.

CASE I.—S. S.——, aged twenty, a nurse by profession. When seventeen years of age she had a severe attack of Southern malarial fever, in consequence of which menstruation was suppressed for eighteen months. Since that time she has suffered with almost constant pain in the pelvis, greatly increased at the menstrual period; she was becoming incapable of attending to her duties, and more and more of an invalid.

The patient called to see her last December. On examination the right ovary was found enlarged, tender, and dislocated down into the Douglas cul-de-sac, and the left ovary was in position and apparently normal. The enlarged and prolapsed ovary was giving her almost continuous distress, and its evidently diseased conditions were affecting her general health and greatly disturbing her nervous system.

On January 20th she performed laparotomy and removed the right ovary and tube; the left ovary seemed all right and in good position, and she was glad to feel justified in leaving it. The incision in the abdominal wall was two and one-quarter inches, length of ovary two and one-half inches, transverse diameter one and three-quarters inch. The patient recovered without a bad symptom. The next day temperature, 90° F.; pulse, 84. Second day, pulse and temperature normal. The operation was to the patient much less trying than would have been the continued treatment, and the results would not have been obtained by it. March 17th, patient was the picture of health, vigorous, contented in mind, and able to work.

In examining the specimen, not a healthy Graafian vesicle nor healthy ovarian tissue was found.

CASE II.—A girl, aged eighteen, came to the dispensary of the Woman's Hospital, pennyless and disheartened; said she had been turned from one situation after another on account of her frequent sickness and consequent inability to work; said her sufferings were indescribable, more than she could bear—pain, sick stomach, and a desire to go to stool when there were no demands of nature. The pain continued all the time, but the agonizing distress commenced some days before menstruation, and continued during the period. She was admitted into the Woman's Hospital of Brooklyn. Her sufferings during the next menstrual period were pitiful.

Examination.—Anteflexion, fundal enlargement of the uterus, and two sensitive masses on each side. Had tried many doctors and much medicine without relief. As soon as patient gained a little strength she removed the uterine appendages under thorough antiseptic precautions, or perfect cleanliness. There were considerable adhesions and some difficulty in removing the appendages. At the end of three days the patient was apparently well, had no more suffering, and now, March 16th, with a face beaming with happiness, she says, "she is glad she came to the hospital." She is now free from suffering and able to work.

The appendages were removed, not because she was suffering, but because they had such a condition of disease as to produce the suffering. Had they been healthy she would not have removed them on account of the great dysmenorrhœa, but would have labored to find out the cause of suffering and attempted to relieve it.

CASE III.—Mrs. L——, aged thirty-six, married, no children; suffered with constant distress in the pelvis for the last thirteen years; great nervous disturbance, prostration, weakness, and inability to attend to her household duties.

Examination.—Ovaries enlarged and exceedingly sensitive, and dislocated low down in Douglas' cul-de-sac. For six months she was treated at the dispensary in hopes of relieving the tenderness, diminishing the size of the organs, and correcting the malposition. No special improvement in any of these respects. After consultation it was decided to remove the uterine appendages. The patient recovered from the operation without a bad symptom, and at the end of the third week she was able to assume the burden of her household duties. March 16th, called at the dispensary clinic, was the picture of health, suffering no distress, and able to work.

CASE IV.—Mrs. M——, aged forty-three, has been suffering for many years; a confirmed invalid, in bed a great portion of the time, subject to sinking spells, at times remained unconscious for three or four hours, complained of great pain in the pelvis and bearing down. For the last five years has had at intervals severe uterine hemorrhages, and during the last five months has had a constant flow without a day's intermission. Examination showed the uterus retroverted and retroflexed, and the fundus enlarged by an intramural fibroid, the appendages extremely sensitive and prolapsed. Laparotomy was performed March 9th. Uterus size of third month of pregnancy, ovaries atrophied, the tubes in a high state of inflammation, the veins of which were converted into great sinuses. At the close of the operation her pulse was 90. She recovered without a bad symptom; pulse and temperature normal.

The appendages were removed, first, because they were diseased; second, to cut off the blood-supply to the fibroid.

CASE V.—Mrs. N——, aged thirty-one, married. The history of the last ten years has been a history of suffering—constant distress in the pelvis and in the back. Examination showed the appendages diseased, sensitive, and prolapsed. When she was taken into the hospital she was literally almost dead. She was kept quiet in bed, had good nourishment, massage, etc. March 14th, laparotomy was performed. April 2d, patient says she feels perfectly well. She recovered from the operation without a bad symptom.

DR. FRANK W. JACKSON presented

A LIVER THAT HAD BEEN THE SEAT OF A GUNSHOT WOUND.

The patient was a male, thirty-six years of age, whose previous state of health had been excellent, except that for two months he had suffered from insomnia and mental depression. One Sunday morning he shot himself with a heavy revolver, carrying a long thirty-two ball, which passed through his white- and under-shirt, both of which it charred, and then entered the abdomen three-fourths of an inch below the tip of the ensiform cartilage, and half an inch to the left of the median line. He was immediately afterward found lying upon the floor. He was assisted to his feet, and he walked a distance of about thirty feet to his bed. He was then seen by a neighboring physician, who probed the wound apparently without antiseptic precautions. His own physician, Dr. Satterlee, saw him half-an-hour later, and found him in good condition, and with very little shock. In the afternoon Dr. Sands passed an antiseptic probe through the wound, three and three-fourths inches, in an antero-posterior direction, showing that the abdominal cavity had been entered. The ball was then found in the back, lying just beneath the skin, four inches below the angle of the scapula and one inch to the left of the spinal column.

THE QUESTION OF LAPAROTOMY

was at once discussed, but in view of the absence of the certainty of a wound of the stomach or intestines, and disinclination of the patient's friends to an operation, it was decided to wait for more definite symptoms. These never came. During the next week the patient's temperature was rarely above ninety-nine and a half, and he had absolutely no symptoms referable to the abdominal cavity. He had a short, hacking cough, and for four days after the injury there were physical signs of consolidation of the lung over an area of about the size of a silver dollar, heard just where the ball was lodged. He twice expectorated a small pellet of bloody mucus.

Seven days after the injury he was allowed to sit up for a while each day.

He did well for two weeks after this, when symptoms of mania came on; he finally became comatose, and after lying in coma for five days he died, just four weeks and four days after the shooting.

The autopsy showed that the peritoneal cavity was free of all evidence of the occurrence of general peritonitis, and there was no evidence that

BLOOD HAD ENTERED THE CAVITY.

Directly over the free border of the liver were found a few fine adhesions. There were also adhesions between the left lobe of the liver and the stomach. The stomach was otherwise intact. The liver showed a scar plainly, and the track of the bullet was traced directly through the substance of the left lobe, from before backward and from below somewhat upward. The ball then passed through the left crus of the diaphragm, and through the posterior mediastinum, but no evidence of wound of the lung was found. There were a few adhesions at the bases of both lungs.

The specimen was presented chiefly to show that the patient *did not die from gunshot wound of the liver*.

Dr. W. H. Porter reported an autopsy in which he found that a bullet had passed directly through the right lobe of the liver, just to the right of the gall-bladder, and there was absolutely no

HEMORRHAGE INTO THE PERITONEAL CAVITY.

The ball passed through the colon, and lodged in the vertebræ. The man lived two or three days after the injury, and died, apparently, from septicæmia.

In another case in which Dr. Porter made the autopsy he found that the bullet had traversed the liver, but there was no hemorrhage into the peritoneal cavity. He had also seen a case of rupture of the liver without hemorrhage.

The Society then went into executive session.

Stated Meeting, April 13, 1887.

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

DR. G. C. FREEBORN presented, in behalf of a candidate, a specimen of *carcinoma of the uterus.*

DR. WACKERHAGEN presented

THE UTERUS AND ITS APPENDAGES EXTIRPATED PER VAGINAM FOR CARCINOMA.

Mrs. H——, thirty-five years of age, a native of the United States, a housewife, and the mother of two children, came under his observation in consultation with Dr. J. E. Gregory, of Brooklyn, March 26, 1887. She had been suffering from pains and hemorrhage, with offensive and irritating discharge, since last December. Her general health had become considerably impaired from loss of sleep and appetite, and she was anxious to take the risk of an operation for the extirpation of her diseased uterus.

The patient was anæsthetized with ether, and, after thorough irrrigation of the vagina and uterus with sublimate solution 1 to 1,000, the uterus was drawn down well with vulsellum forceps in each cervical lip. Two stout silk ligatures were then passed, by means of metallic spindles, through the left and right vault of the vaginal wall, for the purpose of including the uterine artery. An opening into Douglas' cul-de-sac, which was made with scissors (blunt), permitted the introduction of a finger into the peritoneal cavity, for the proper placing of the succeeding ligatures, but before these were applied the general peritoneal cavity was protected by an antiseptic sponge, and the mucous membrane and peritoneum covering the distal portion of the stump were sewn together by a continuous catgut suture, this last manœuvre effectually controlling hemorrhage. With a sound introduced into the bladder, a similar opening was made anteriorly. Here, also, the peritoneum was everted by a tenaculum and united to the vaginal wall by a continuous suture.

The left index-finger being now carried across and behind the left lateral mass between the two openings penetrating the peritoneum, this tissue was tied off in five sections. Attempting to carry out the same plan on the right side, it was found that the anterior incision had been too short and the finger could not pass so large a mass, to guide the ligating of this right vaginal wall, and consequently the ligation was made from both extremities toward a common point. This accomplished, the whole vaginal vault had been securely ligated and separated from its uterine attachments, and hemorrhage stopped without the loss of more than an ounce of blood.

The fundus uteri was now turned out and the ovaries brought down. There remained attached a portion of both broad ligaments on each side. These were tied off with silk, cut, and the uterus and its appendages laid aside. A portion of one broad ligament, and a small mass of the posterior stump oozed a little blood and demanded a secondary ligation. The stump was then stitched to the vaginal wall in several places to prevent its retracting and becoming inaccessible in the event of secondary hemorrhage. After thorough cleansing internally and externally and removing the sponges, which had very effectually shielded the peritoneal cavity (for their innermost surfaces were scarcely discolored), iodoform dressings were applied and the patient placed in bed. Her pulse was remarkably quiet and natural, and consciousness was restored within a short time. No vomiting occurred, and up to the present writing the temperature had not been above $99\frac{2}{5}$° F. Dr. Wackerhagen was under obligations to Dr. Paul F. Mundé, who kindly assisted him in the operation.

The specimen was a multiparous hyperplastic uterus, cut longitudinally through the posterior wall, and with a tributary cut extending into the right cornua. The body was four inches long, with walls showing an excess of fibrous elements. The major portion of the enlargement was in the cervix, and of this particularly the right half of the anterior lip. By reason of this asymmetry the os externum did not form the apex of the irregular excavation in the invaginated portion, but was fully seven-eighths of an inch lower and five-eighths of an inch farther to the left. The mucous membrane was hyperæmic and thickened. The right ovary was cystic.

The Society then went into executive session.

Stated Meeting, April 27, 1887.

T. MITCHELL PRUDDEN, PRESIDENT, IN THE CHAIR.

DR. FREDERICK PETERSON (Poughkeepsie) presented portions of lungs from a case of

ANTHRACOSIS PULMONUM.

G. R——, sixty-seven years of age, and a worker in the cleaning-room of a stove foundry, fell dead in the street. Dr. Peterson was sent by the coroner to make a post-mortem examination, which took place five hours after death. The body was well formed and well nourished. *Rigor mortis* absent. The pupils were dilated. There was nothing abnormal about the brain.

Thorax: On opening this cavity both lungs were found bound to the chest-wall by old and strong adhesions. They were extraordinarily black over the whole surface, and strikingly hard and lobulated (very much like a cirrhotic liver, except that the nodules were larger). There were numerous patches of emphysema, the dilated and confluent air-vesicles being in places the size of peas. There was œdema to a great degree in both lungs. In the left pleural sac was about a litre of clear serum. The whole upper lobe of the right lung was one extremely hard tumor, like a fibroma, and perfectly black on cutting through it. In this tumor was a small abscess the size of a hazel-nut. When the hands were brought into contact with a freshly cut lung-surface, they became discolored and blackened, as if one were handling bone-black with wet fingers. The bronchi, bronchial tubes, and trachea had clean, white mucosæ. The mediastinal glands were greatly hypertrophied, wholly black with carbon, and very hard. The pericardium contained sixty cubic centimetres of clear serum. The endocardium was exceedingly fatty, somewhat hypertrophied, and dilated. This fatty metamorphosis was, of course, due to the condition of the lungs. The mitral, pulmonary, and aortic valves were somewhat thickened, and at the bases of the pulmonary and aortic lunulæ were small calcified patches, but not sufficiently large to produce stenosis of the orifices. There was an advanced general endarteritis deformans.

Abdominal cavity: The *spleen* was a little enlarged, hard, and very adherent to the diaphragm. The *liver* was small, very fatty, and everywhere strongly adherent to the diaphragm. There was chronic *gastric* catarrh. Both kidneys showed signs of senile atrophy. All the other organs were normal.

A certificate of death from fatty degeneration of the heart was made out. The most interesting pathological objects were the lungs, with their enormous quantities of carbonaceous matter, and also in the lymphatic glands in their neighborhood. Undoubtedly this carbon had caused fibrous phthisis, the pleuræ and peritoneum on the diaphragm subsequently becoming involved.

This man had worked in the cleaning-room of the stove works for thirty-one years. During all this period he had at no time manifested physical illness other than a "terrible morning cough," as his wife termed it. The cough he had had always while at this occupation. In this cleaning room is brushed from the iron castings the charcoal dust which is used in their manufacture. The atmosphere of the room is so dense with carbon that one can scarcely see through it, and it is nearly irrespirable to one unaccustomed to it. There is also a small amount of sand in the air. ALL OF THE LARGE FORCE OF MEN EMPLOYED IN THIS ROOM SUFFERED FROM PULMONARY DIFFICULTIES. The case was interesting because it showed what devastation this occupation could cause in the lungs, and yet how assiduously one could pursue it without apparent shortening of his life. It was probable that this particular man had a fine constitution. But many others had spent a decade or more in the same room without further detriment than the bronchial catarrh and cough.

DR. IRA VAN GIESON presented a

KIDNEY CONTAINING A LARGE CALCULUS.

The main part of the stone was cylindrical, *five* centimetres long and one centimetre in diameter, and was lodged in the sinus of the organ, with two prolongations, each about one centimetre long, entering into calyces. There were indentations of the surface of the kidney corresponding to the position of the branches of the calculus in the calyces.

DR. VAN GIESON also presented specimens illustrating the lesions of

PHOSPHORUS-POISONING,

death occurring on the fourth day.

A middle-aged man, on February 1, 1887, dissolved a box of ordinary phosphorus matches in a glass of water and drank it. He entered Bellevue Hospital twenty-four hours afterward, where he was given tea, which he immediately vomited. He continued to vomit all medicine which was given him and died, February 4th, at noon, with slow respiration and jaundice.

Autopsy, twenty-four hours after death. The *heart* showed grayish-white patches near the ventricles; the valves were normal. Old atheroma in the aorta; small ecchymoses beneath the diaphragmatic pleura. *Lungs* œdematous and congested; *bronchi* trabeculated, and contain muco-pus. *Liver* enlarged, fatty, with slight increase of interstitial connective tissue. *Kidneys*. The capsules were adherent, the cortical markings distinct, the labyrinths grayish-white in color, and looked swollen. *Spleen* slightly enlarged. In the *stomach*, near the upper curvature and near the pylorus, there was an oval patch of erosion of the mucosa about three centimetres long; there were also smaller scattered erosions. In places the mucosa had a dull, grayish-white, thickened appearance. The mucosa of the *duodenum*, for a short distance beyond the pylorus, showed the same appearance, but there were no erosions. The *small intestine* and Peyer's patches showed chronic congestion. In the *trachea* the mucosa was covered with closely aggregated translucent elevations, looking like

MILIARY TUBERCLES.

These ceased at the bifurcation. At the bifurcation of the primary bronchi there were a number of deep pin-point erosions. The *œsophagus*, near pylorus, was covered with a brownish adherent film, and there were several small sharply defined erosions of the mucosa. Several of the cervical lymph-glands were of a deep-red color. The *mesenteric glands* were normal.

Microscopical examination.—In the *heart* fatty degeneration; the *lungs* showed the lesions of heart disease. In the *liver* the cells were swollen and contained fat droplets. The *kidneys* showed the lesions of parenchymatous degeneration. In places in the *stomach* the mucosa was nearly of normal thickness; in other places the thickness was much decreased; in places the principal cells of the follicles were swollen and granular, and completely filled the lumen of the follicle; in other places the principal cells were few in number, and the peptic cells were very numerous. With osmic-acid staining both varieties of cells showed minute fat-droplets. The epithelia of *Brunner's glands* were swollen and granular—as were the cells of the *pancreas* and the mucous glands of the trachea. It was the swelling of the latter cells which caused the prominent elevations of the mucosa. Sections from one of the bronchial glands showed miliary tubercles. The deep-red, cervical lymph-nodes showed a distention of the peri-follicular spaces and the lymph-sinuses of the medulla of the node, with red blood-cells, and there was a notable decrease in the number of small round cells in these nodes. One of the bronchial glands exhibited the same appearance.

Remarks.—The following were the interesting features of the case: First,

THE VERY SELECT AREA OCCUPIED BY MILIARY TUBERCLES—NAMELY, ONLY ONE BRONCHIAL GLAND.

This showed one of the stages in the development of tuberculosis, and illustrated that constant breathing of tubercle-bacilli might, in some, set up tuberculosis of the bronchial glands. The second point of interest was the peculiar appearance presented by some of the cervical glands, as if a blood-vessel had ruptured into some of the lymph-channels, or the blood had been conducted into these glands and had lodged into the peri-follicular spaces and lymph-sinuses of the medullary portion. Specimens were exhibited under the microscope.

The stomach was interesting on account of the *increased number* of *peptic cells*, probably due to the stimulating effect of the phosphorus, and a conservative effort to supply the place of those that had been destroyed by the poison.

The President remarked that the specimens presented by Dr. Van Gieson showed remarkably well the lesions which had been described, and he regarded them as especially interesting in connection with the question of the absorption of pigment in the lungs, shown to such a remarkable degree in the specimens presented by Dr. Peterson.

LARYNGEAL, TRACHEAL, AND BRONCHIAL DIPHTHERIA—INTUBATION.

Dr. A. Brothers presented specimens from a case of diphtheria with the following history:

F——, aged nineteen months, was seen first on the morning of April 5, 1887. The boy had been suddenly taken sick during the previous night with symptoms of croup. At his first visit the temperature was 103° F. and respiration 35. The cough and breathing were distinctly laryngeal. There was moderate dyspnœa, with dilated nostrils. Dr. F. Huber, who saw the case at this time, expressed it as his opinion that the dyspnœa was in part pulmonary. An examination of the lungs showed dulness over the upper portion of the right lung. By night the difficulty in breathing had become decidedly worse.

THE THROAT SHOWED CONSIDERABLE MUCOUS DISCHARGE, BUT NO FALSE MEMBRANE.

There was marked retraction visible in the supraclavicular and epigastric regions with each inspiration.

The lips were beginning to be cyanotic. In view of the urgency of the symptoms, Dr. Brothers decided to intubate, and succeeded, after several attempts, in introducing the O'Dwyer tube. This was followed by the characteristic cough and immediate abatement of the dyspnœa. On the following day the temperature was 101° F. and breathing 35 per minute. The child had not slept much all night. On the 7th the child was decidedly worse. The temperature was 102½° F. and respiration 50. The dulness over the right lung had increased. On the 8th the temperature was 103½° F. and respiration 60 It was now impossible to nourish the child, on account of the severe paroxysms of cough caused by each attempt to swallow. During the night the child became worse, and died on the morning of the 9th—the fourth day after intubation. The treatment had consisted of bichloride of mercury (one-fourth grain in the twenty-four hours), brandy, antipyretics (antifebrin and antipyrin), and steam inhalations.

Autopsy.—This was made five hours after death, with the assistance of Dr. A. T. Joyce. An incision in the neck only was permitted. The portion of tonsils and pharynx removed presented no false membrane. The larynx with its contained tube was taken out, and showed the latter to be in proper position. The arytenoid cartilages were slightly eroded at those points which had served as support for the shoulder of the tube. The lower end of the tube was not obstructed by membrane. Its cavity contained a gelatinous material, probably a mixture of mucous and membranous débris. The larynx, trachea, and larger and smaller bronchi were lined by false membrane which could be peeled off. The lower lobe of the right lung was completely hepatized, so that a section of it in water at once sank to the bottom. The apex of the left lung was in a similar condition. There were no pleuritic adhesions. The other organs were not examined.

Dr. Brothers made a very careful examination of the smaller bronchi, but could not find evidence that any particles of food, after intubation was practised, entered the lungs. The epiglottis covered the tube, and there was evidence of pulmonary consolidation before the tube was introduced. The case did not speak against intubation, because there was evidence that false membrane was present in the smaller bronchial tubes before the tube was introduced, thus putting it in the class of cases in which the disease is almost always fatal.

DR. FRANK FERGUSON presented a

FRESH SET OF SPECIMENS.

They were interesting on account of their freshness and the multiple lesions which they illustrated. They were taken from the body of a man forty-eight years of age, a native of Ireland, and a trunkmaker by occupation. He was admitted into the service of Dr. George P. Wheelock, attending physician to St. Luke's Hospital, on April 26, 1887, to whom he was indebted for the clinical notes and the opportunity to present the material. The patient never had rheumatism. When three years of age he had scarlet fever. His family history was negative. For a long time he used alcohol freely, taking it often before breakfast.

For three years previous to his admission into the hospital he had swelling of his feet and lower part of the abdomen, lumbar pain, headache, visual disturbances, and dizziness.

Three weeks before his death his feet, scrotum, and abdomen became greatly swollen, which gradually increased until his death. He suffered greatly from dyspnœa, he became delirious, and died twenty-four hours after admission. He said at the hospital that he had passed no urine for four days, and no fæces for five days.

Autopsy.—There were one hundred and twenty ounces of serum in the peritoneal cavity, and a few ounces in each pleural cavity. There were four ounces of serum in the pericardium. The *heart* was large and soft, and its muscular tissue contained fat. The valves were competent. The *lungs* were congested and œdematous. The *spleen* was four times its normal size, and very soft. The *stomach* was very intensely congested. The mucous membrane was thickened, but its muscular wall was normal. The *kidneys* were firm in consistency, the capsules were not adherent, the surfaces were smooth, and the markings of cortex were quite distinct. There was fat in the straight tubes. The *liver* contained a very great quantity of fibrous tissue and fat. The *pancreas* was fatty and very firm in consistency. It was slightly larger than normal.

The Society then went into executive session.

Stated Meeting, May 11, 1887.

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

MEASLES FOLLOWED BY PNEUMONIA.

DR. W. P. NORTHRUP presented specimens removed from the body of a child that died in the New York Foundling Asylum, and for the clinical history of the case he was indebted to Dr. Meyers, house-physician. The child was brought in on May 3d, sick with measles, and it died of pneumonia on May 11th. There was only slight disturbance of respiration, and no symptoms of croup. What dyspnœa existed was supposed to be due to well-marked double pneumonia.

At the autopsy, which was performed very early, there was found false membrane from the tip of the nose to the terminal portion of the finest bronchi. In the larynx there was abundance of false membrane, and yet

THERE WAS NO EVIDENCE OF CROUP.

This point was interesting in connection with cases of measles which he had seen with croupy cough, and yet no false membrane was found. But, at the anterior commissure of the vocal cords there was found a small ulcer, which showed a slight amount of fibrin in the slough. If that was diphtheria, it was the only spot where it existed. In the measles cases there was marked croupy cough and loss of voice. In this case there was neither loss of voice nor symptoms of croup, and it was not suspected that there was any exudation in the larynx. The false membrane was also abundant in the trachea, and it could be distinctly traced into the lungs as a thick, tough membrane. The lower lobe of the left lung was nearly solid, and that of the right one presented the same appearance, and corresponded to the condition described as collapse. But he inflated the right lung, and the inflation was easily made complete. This had been given as

ONE OF THE CRUDE TESTS FOR ATELECTASIS,

and therefore, according to the test, the condition present should be that of atelectasis, and not of pneumonia. In no case, however, of diphtheria of the trachea and bronchi, where the lungs showed these elevated and depressed areas upon the cut surface, had he failed to find

EVIDENCE OF PARENCHYMATOUS PNEUMONITIS.

That is, in the alveoli were found pus, swollen epithelia, and occasionally fibrin.

This child was sick, from the third until the eleventh day of May, with measles and pneumonia. Within half an hour after death he drew urine from its bladder, and found that it contained albumen. It also contained casts in abundance, and the kidney was found to be the seat of parenchymatous nephritis. The kidneys were exceedingly anæmic—so markedly so that he suspected the pale appearance might be due to the rapid exsanguination caused by making the post-mortem so early. The lungs, also, were anæmic, except where the pneumonia existed. This he had seen before, but had never seen the peculiar pallor of the kidneys.

Dr. Northrup also presented specimens from a case of

GENERAL TUBERCULOSIS WITH HEMORRHAGES IN THE FLOOR OF THE LATERAL VENTRICLES, AND DILATED TUBERCULOUS BILE-DUCTS.

The child was aged two and a half years. No diagnosis was made. The complaint was that, on the day previous, it vomited. It was admitted at ten o'clock, had eleven convulsions, and died the next morning at 8.30 o'clock, never having recovered consciousness.

At the autopsy the cerebral convolutions were found very much flattened, the ventricles dilated and containing from four to six ounces of fluid, and on the floor of the lateral ventricles, beneath the ependyma, were found minute punctate hemorrhages. There were tubercles upon the ependyma and at the base of the brain. There were tubercles in the lungs, without consolidation.

The *liver* was very much enlarged, the bile-ducts were dilated, stained with bile, and had thickened walls surrounded by diffuse tubercular tissue. He had not seen this condition of the bile-ducts in adults, but he believed that it was present in the majority of cases of extensive tuberculosis in children.

Dr. Heineman remarked that the portions of lung involved in pneumonia in *children* were frequently below the level of the cut surface, which was one of the striking differences between the appearance of pneumonia in children and that of adults, where the portions involved were frequently *above* the level of the rest of the surface; that is, above the portions containing air.

Portions of these lungs become collapsed and settle below the level of the surrounding parts containing air, because the bronchi are involved and prevent expansion of these particular groups of acini; so that the collapse is simply an accident, and not of much importance, although it had been made much of. He was unable to see why the test should ever have been brought forward; that if a lung could not be inflated it was pneumonic, whereas a pneumonic lung could be inflated. If a little mucous plug at the terminal part of a fine bronchial tube could give rise to feeble respiration and prevent proper inflation during life, he was unable to see why a slightly increased force could not inflate it after death, as had been done in these lungs presented by Dr. Northrup, by applying a comparatively slight degree of force.

As to the anæmic condition of the kidney, he doubted if the mere fact of emptying the organs of blood by a sudden gush from the divided aorta had anything to do with the gross appearance which required the microscope for its solution.

DR. CHEESMAN had seen the same pale appearance of the kidneys of rabbits that had been bled to death rapidly.

THE PRESIDENT fancied that a good deal that had heretofore been said concerning the color of organs after death would have to be revised. He would ask Dr. Northrup if he had noticed any of the karyokinetic figures in his search for evidence that the new cells were produced from the epithelia of the alveoli.

DR. NORTHRUP said that he did not. He would inquire if this condition of the gall-ducts had been noticed in adults.

DR. HEINEMAN remarked that tubercle of the gall-ducts in adults was not common; perhaps for the same reason that in general tuberculosis, tubercles were usually very abundant in the liver of children, whereas in the adult liver only a few were found.

The Society then adjourned.

Stated Meeting, May 25, 1887.

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

DR. JOSEPH W. HOWE presented a patient on whom he had

LIGATED THE FEMORAL ARTERY UNDER COCAINE—SECONDARY HEMORRHAGE — TRANSFUSION OF LITTLE'S SOLUTION—RECOVERY.

The history of the case was as follows :

William B——, aged thirty, occupation fish dealer, was admitted to St. Francis Hospital January 18, 1887. He uses stimulants freely, and has had gonorrhœa and syphilis.

Six months ago he noticed a swelling in the popliteal space on the right side, which gradually increased in size and for four months past has been extremely painful. On examination a large pulsating tumor was found to occupy the whole of the popliteal space on the right side, extending upward to Hunter's canal. Over the tumor a loud rasping bruit was audible.

Two large angular sloughs were found in Scarpa's triangle, due to compression with the tourniquet before he entered the hospital.

On January 26th he injected a four per cent. solution of cocaine over the site of incision and ligated the femoral artery in the lower part of Scarpa's space, without pain. During the operation, however, the patient became pale and pulseless, and the operation had to be suspended for a few moments in order to administer stimulants. This collapse, he thought, was due to the cocaine.

When the operation was completed the leg was wrapped in cotton and a dose of opium and quinine given Knowing that the previous attempt to cure the aneurism by compression had produced much sloughing, and being fearful of secondary hemorrhage, he took the precaution to keep a member of tne house staff with him, and to place a tourniquet around the limb, ready to be tightened at a moment's notice.

On February 10th, fifteen days after the operation, there was considerable oozing of blood from the lower part of the wound around the ligature, which was stopped by making pressure with the tourniquet.

On the night of the 11th, the tourniquet being loose, a sudden gush of blood took place, which was stopped with the tourniquet by the house surgeon, Dr. Spence, but not before a large quantity of blood had been lost. On February 12th something went wrong with the tourniquet again, and another severe hemorrhage took place, and although the tourniquet was rapidly tightened, the patient was exsanguinated before the flow of blood ceased. The pulse could scarcely be felt at the wrist, there was great thirst which was not relieved by liquids. There were also great restlessness and sleeplessness. The day following Dr. Howe concluded to perform transfusion, and not being able to get blood to transfuse, he injected into the cephalic vein ten ounces of Little's solution, prepared by the house physician, Dr. Hodenpyl. There was some relief from the collapse by the operation, but on the day following he seemed to be as low as ever, and he opened the same vein and injected *sixteen* ounces of the same solution. This latter injection was followed by immediate and well-marked relief. The pulse became fuller and the voice stronger. The pain in the lumbar region disappeared, and the patient expressed himself as feeling first-rate. There was no doubt about the efficacy of the injection.

The members of the house staff watched the patient faithfully night and day, keeping the tourniquet applied for a period of three weeks. At the end of that time the instrument was removed and no more hemorrhage occurred.

The patient's condition at this time, March 3d, was not good. The leg was œdematous and purple. The knee-joint was filled with liquid, and he was still very weak. He, however, gradually gained flesh and strength, and was discharged from the hospital in May.

There were two interesting points to consider in connection with this case, viz., the origin of the hemorrhage and the success attending the transfusion of nearly two pints of a saline solution instead of blood. The flow of blood was such as could only come from a very large artery. Query: Did it come from the femoral artery? He thought it did. Dr. Shrady, his colleague, thought otherwise, especially because there was no recorded case of recovery after any such accident. If it did not come from the femoral artery, where else did it come from? The blood started at the site of the ligature on the fifteenth day. The patient was exsanguinated by a few seconds' flow.

Then, with regard to the injection of the saline solution, he must say he had no faith whatever in it. He had never employed it before, and he only used it then because blood could not be had. The second transfusion, he thought, without doubt saved the patient's life.

Some time afterward he injected twelve ounces of the same solution into the veins of a boy in the hospital who had phthisis, Pott's disease, and abscess. It did him no good.

Jennings' Solution.—℞. Sodii chlor., gr. l.; potass. chlor., gr. iij.; sodii sulph., and sodii carb., āā gr. ijss.; sodii phosph., gr. ij.; alcoholis, ʒ ij.; aq. dest., O j. M. et S.: Inject at 100° F.

Little's Solution.—℞. Sodii chlor., ʒ j.; potass. chlor., gr., vj.; sodii phosph., gr. iij.; sodii carb., gr. xx.; alcoholis, ʒ ij.; aq. dest., ℥ xx. M. et S.: Inject at 100° F.

Dr. Howe also presented a specimen removed from a case of femoral aneurism, in which he

LIGATED THE EXTERNAL ILIAC ARTERY UNDER COCAINE.

It was accompanied by the following history:

John W——, aged twenty-one, single, of Irish birth, and a boilermaker, entered the hospital January 18, 1887, complaining of severe pain in the right leg, and especially along the inner side of the thigh. It had begun five days before this, and was accompanied by numbness of the limb and severe pain on attempting to walk.

In September of the previous year he had suffered from an attack of typhoid fever. He had also had several severe attacks of acute articular rheumatism, and for four years believed his heart to have been affected, although he had never suffered from it further than from slight dyspnœa on exertion.

There were no evidences of venereal disease, and no history of alcoholic excess.

On examination the heart was found to be acting strongly; the pulse-rate was about 90. There was a loud mitral regurgitant murmur, and also an aortic regurgitant. The lungs and the abdominal organs showed nothing abnormal. The urine was examined with negative results. Locally, nothing was found to account for the pain. The patient was in a fair general condition, although somewhat anæmic. There was a slight elevation of temperature daily.

On the 25th, the second and third toes were found to be livid, and the whole extremity was cold and somewhat swollen.

On February 1st, a slight swelling was noticed immediately below Poupart's ligament in the line of the femoral artery, and this rapidly increased in size. By the 15th, there was a swelling the size of a hen's egg, which showed pulsation and presented a well-marked bruit. Pressure could not be applied below Poupart's ligament, and was very badly tolerated above.

The circulation had improved in the leg, and there was less tendency to gangrene. The patient's general condition had grown worse, and morphine had to be freely used to secure relief from the pain. He was then transferred to the surgical side of the hospital for treatment. The aneurism still continued to increase very rapidly in size, and on February 27th, scarcely four weeks from its first appearance, the skin over it was livid and softened, and rupture of the sac seemed imminent. Without hope of permanently benefiting the patient, but simply to do away with the danger of sudden death from rupture of the sac, he ligated the external iliac artery, first, however, injecting into the subcutaneous cellular tissue, over the artery, fifteen minims of a four per cent. solution of cocaine. This quantity of cocaine sufficed to make the operation a painless one, excepting at its termination, when the edges of the wound had to be widely separated to get at the artery.

On the third day after operation there was free oozing from the wound, which was opened and a search made for bleeding points. Slight compression was all that was necessary, however, to control it. On the sixth the temperature rose to 104½, and the abdomen became swollen and painful. The ligature came away on the ninth. He grew weaker rapidly, and on the tenth he died.

The autopsy showed the heart to be hypertrophied, and the aortic and mitral valves roughened and covered with vegetations. The pericardial sac contained a large amount of fluid. The lungs were congested. The intestines were distended. In the right iliac region, near the seat of operation, there were some fibrinous adhesions. There was no effusion. Both extremities of the ligated artery showed firm clots.

The points of interest in this case are first, the rapid development of a femoral aneurism in a young, bed-ridden patient; and second, the successful performance of the operation, without pain, by the use of cocaine as a local anæsthetic.

In this connection he wished to state that he had also amputated the mammary gland for scirrhus, in St. Francis Hospital, using during the whole operation forty minims of a four per cent. solution of cocaine. The operation was completed without pain, but at its termination the patient vomited. Whether this was the result of the cocaine or from shock, he was not prepared to say. These are the first cases of the kind on record where cocaine has been used instead of ether or chloroform.

Dr. Wyeth remarked, with regard to Dr. Howe's first case, that it was very unusual for constitutional effects to be developed by the injection of twenty minims of a four per cent. solution of cocaine, and he made this remark because he was so much in favor of the use of cocaine in surgery that he would dislike to have it discarded through fear to employ it. To be sure, it was not impossible for the effect mentioned by Dr. Howe to occur as a consequence of this moderate quantity, but as he had used the drug very extensively for the purpose of producing local anæsthesia, and had seen its unpleasant constitutional effects produced in only one case, and that where a drachm of this solution became actually forced into the tissues, he believed that the cases in which such effects would be produced were very rare.

With regard to

TRANSFUSION OF A SALINE SOLUTION

in Dr. Howe's first case, Dr. Wyeth said he was glad that Dr. Howe had acknowledged his conversion to the use of saline solutions for this purpose. He had had two cases in which he had practised injection of this solution with very marked benefit. One occurred in the practice of Dr. Sims, and the woman became exsanguinated in labor before the doctor's arrival. In that case, Dr. Wyeth injected twelve ounces of Little's solution into the median cephalic vein ; the improvement was immediate, and the patient recovered. It was Dr. Sims' opinion that she would have died, had she not had the benefit of the transfusion. The second case was one of hemorrhage after ovariotomy, in the practice of Dr. Mundé. In that instance twelve ounces of the saline solution were injected at two different times, and both injections improved the condition of the patient immediately and very markedly ; but, despite the benefit, the case terminated fatally.

Dr. Wyeth thought it was now generally accepted by surgeons that there was no better agent for transfusion than the saline solution.

Dr. Howe was unwilling to go so far as Dr. Wyeth in commending the saline solution, for he still believed that

THERE IS NOTHING BETTER THAN BLOOD FOR TRANSFUSION,

and that the saline solution was next best, and that it answered an admirable purpose where blood could not be obtained. Besides, to use the blood of an excited, nervous relative for transfusion was very unfavorable.

Dr. Wyeth presented

A BULLET

thirty-two calibre (Smith & Wesson's), removed from a man in whom it had lain for three and a half years. It passed through, and was followed by collapse and loss of function of, the right lung. It entered the third intercostal space, and lodged in the scapular region. The operation for its removal was performed under cocaine anæsthesia.

The case was made the text for the report of one of

STAB-WOUND OF THE LUNG,

that came under his care one year ago, in a man twenty-eight years of age. He was stabbed five times, and three times the blade entered the left lung from the middle up to the apex. Profuse hemorrhage followed from the wounds and also from the mouth. Collapse of the lung and empyema followed, for which Dr. Hunter McGuire, of Richmond, operated three times by incision into the thorax and drawing off the pus. The patient gradually failed, however, and finally came to this city, when Dr. Wyeth found a circumscribed empyema six by four inches and two inches deep. After the patient was anæsthetized he found that the incisions made by Dr. McGuire failed to give complete relief, because the ribs closed the opening almost immediately, and prevented drainage. Dr. Wyeth excised two inches of one rib, and then carried in a stiff drainage-tube and drained the cavity from the bottom. The patient improved, and, in the course of six months, recovered. The case was interesting because it showed the

NECESSITY OF SECURING PERFECT DRAINAGE.

The cavity was irrigated by varying strengths of a solution of bichloride of mercury, 1 to 3,000, 1 to 1,000, and even 1 to 500; and at times he felt that he was subjecting the patient to considerable risk, but no unpleasant results followed. He used the strong solution for the purpose of killing the pyogenic property of the lining membrane of the abscess cavity. In the Surgical Society stress had been laid on the absorption of a solution of 1 to 5,000 by the flaps after amputation at the hip-joint, so as to produce toxic effects, which he regarded as impossible, and he brought this case up in comparison.

THE PRESIDENT said he had often wondered, in looking at pyogenic membranes, which are so distinctly septic, how either weak or strong solutions could be efficacious, because the colonies of micrococci were often so deep in the tissues.

DR. WYETH said that it was with the view to reaching the deep-seated colonies that he curetted the surface with his finger-nail, and practically hyper-distended the cavity.

DR. PUTNAM-JACOBI suggested that it was not really the antiseptic, but the irritant, action of the strong solution that was obtained.

Dr. Van Santvoord presented

THE EAR AND TEMPORAL BONE

removed from a child aged eighteen months, that had had middle-ear disease of long standing, and died apparently from exhaustion, with marked emaciation.

On opening the skull a small abscess was found under the arachnoid at the root of the seventh nerve, on the left side. There was a sub-dural abscess over the petrous portion of the temporal bone. On opening the ear it was also found that the bone had been formed under the temporal fascia, and that the whole of the petrous portion was necrotic.

The point of special interest was the situation of the abscess with reference to an incision for its evacuation. The usual rule is to make the incision one-fourth of an inch, or perhaps more, behind the ear, but in this case such an opening would have been of no avail, as it was necessary

TO MAKE THE CUT DIRECTLY IN THE CREASE BEHIND THE EAR AND FORWARD.

He regarded this as an important point in connection with the surgical procedure adopted for giving exit to pus in these cases.

Dr. Garrish thought it was proper to trephine in all these cases, when there was evidence of severe inflammatory action.

Dr. Putnam-Jacobi presented a specimen of

DERMOID CYST BY INCLUSION,

removed after death from the body of a woman forty years of age, and the mother of nine children, who was admitted to the infirmary February 11, 1887. She said that her abdomen had been enlarged eighteen years, but that it had not increased in size during the last three years. She had been able to work until three weeks before admission, when she felt that something had ruptured in her abdomen.

On admission, there was found an immense tumor, reaching up to the epigastrium, that had separated the recti muscles, and allowed an umbilical hernia to form. The posterior wall of the vagina was prolapsed, and the cervix uteri was crowded under the symphysis pubis. The circumference of the abdomen at the umbilicus was 58½ inches; the distance from the umbilicus to the pubis, 19 inches; from the umbilicus to the ensiform cartilage, 18 inches; and from the umbilicus to the anterior superior spinous process of the ilium on either side, 19½ inches. The patient failed rapidly and died.

At the autopsy, the muscular walls of the abdomen were found atrophied, and adherent to the peritoneum. This was greatly thickened, and closely adherent to the tumor, and the cavity contained an amber-colored, gelatinous substance, together with much yellow fluid. The tumor was found on the left side, and below the umbilicus. The sac had a large opening with smooth edges at the upper part. At this point the cyst had evidently ruptured at some time, and allowed the escape of the fluid and gelatinous material which filled the abdomen above it. Four or five masses, about the size of hen's eggs, were found attached to the omentum above the level of the tumor, and contained sebaceous matter and hair. The question arose, whether or not the rupture occurred so late as six weeks before death, when the patient had suffered from a sudden pain in the abdomen, and sensation of something bursting inside? There was then a distinct cyst within the peritoneal cavity to which the peritoneum had become closely adherent. The left ovary could not be found. At the outer part of the tumor the coils of intestine were so closely adherent, and the adhesions were so numerous, that it seemed remarkable that the woman had not suffered from symptoms of intestinal obstruction. The cavity of the tumor was filled with amber-colored, semisolid fluid, and masses similar to those found attached to the omentum. At one place spiculæ of bone could be distinctly felt, and a portion of a maxillary bone with one well-formed tooth were found. The masses within the sac also contained hair and sebaceous matter. The tumor seemed due to inclusion of germs from a twin embryo, rather than a degeneration of an ovary; for it was only connected with the pelvic organs by a fold of peritoneum, which passed from its lower surface to the upper or posterior wall of the uterus.

The President presented a specimen of

THROMBOSIS OF THE RIGHT AURICLE WITH CENTRAL DEGENERATION OF THE CLOT.

A German bartender was admitted to hospital April 8th. He answered questions reluctantly, and the previous history was therefore incomplete. He was a heavy drinker, and had been jaundiced for five years. There had been no vomiting. He had been in bed for three weeks, and for two weeks had been short of breath. His right arm was swollen, but he would not say how long it had been so. He was considerably jaundiced on admission, well-nourished, his mouth dry and brown with sordes. Respirations were thirty-two; chest expansion, fair. Marked dulness and tubular breathing at right apex, and voice much increased. No râles. Left apex breathing and voice increased.

Heart-apex fifth space, three inches from median line, regular, fair force, no murmur. There was marked subcutaneous œdema of the right arm and hand from neck downward. Nothing abnormal could be detected in the veins of the arm. There was a little œdema in the under-part of left arm and forearm, but none in shoulder or hand.

Pulse ninety-six, feeble, regular, rather high tension. He was in a state of low delirium, and died on the day following admission.

Autopsy.—Right auricle and ventricles moderately dilated. Valves normal. In the right auricle, just beside the entrance of the descending vena cava, and somewhat encroaching upon it, was a white, smooth-surfaced, globular, projecting thrombus, about one inch by three-fourths inch in diameter, closely adherent to the auricle wall by a broad base which quite filled up the cavity of the auricle.

The wall of the auricle at the seat of adhesion of the thrombus was rough on the inner surface, and the seat of several small areas of necrosis, while the muscle fibres in this vicinity were in places necrotic, and markedly fatty. The central part of the thrombus was softened and formed a whitish semi-fluid mass which had the gross appearance of pus.

The central softening was so extensive that in places a thin wall only remained, enclosing the detritus. This softened material was found, microscopically, to consist of fragments of red and white blood-cells—the latter mostly fatty—and granular and fatty detritus.

Aside from a large central area of softening, the thrombus showed similar smaller areas in these parts, which were entangled among the projecting muscle-bundles of the auricle.

The subclavian and jugular and the veins of the arm appeared normal, as did the larger vessels of the lungs. The upper half of the right upper lobe of the *lungs* was the seat of excessive congestion and œdema. The posterior half of the lower lobe showed moderate congestion and œdema. The left lung showed moderate congestion and œdema of the posterior part of both lobes. There was general chronic bronchitis. The *liver* showed chronic congestion with chronic interstitial inflammation, both in moderate degree. The gall-ducts were patulous, the gall-bladder empty. *Kidneys* normal.

The thrombosis appears to have been secondary to a localized endo- and myocarditis of the auricle, of a necrotic form. The cause of the apparently primary lesion, he was unable to state.

He presented the specimen chiefly because it was so excellent an example of the lesion which has become classical under the name of the "globular thrombus" of Laënnec, and to show how thin and fragile may be the still firm portion of such a clot, which is all that retards the occurrence of multiple embolisms, and an increased complexity in clinical symptoms already obscure.

DR. J. LEWIS SMITH presented, in behalf of Dr. W. P. Northrup, specimens from a case of

MEASLES COMPLICATED BY BRONCHO-PNEUMONIA AND VERY MARKED PARENCHYMATOUS NEPHRITIS,

occurring in a child aged two years and three months.

DR. GARRISH exhibited microscopic sections of

A DISEASED KIDNEY OF THE HORSE.

The Society then went into executive session.

Stated Meeting, June 8, 1887,

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

DR. PUTNAM-JACOBI presented, in behalf of a candidate, a specimen of *acute atrophy of the stomach, with fatty degeneration of the heart.*

DR. DUDLEY presented

THE UTERINE APPENDAGES

in the case of an unmarried woman, aged twenty-two, with the following history : At the age of six years she had had small-pox, from which she made a good recovery ; from six to twelve years of age she had some of the exanthemata and diphtheria ; at the age of twelve she showed nervous symptoms, and would occasionally faint, especially if in a crowd ; at times she complained of headache ; at maturity her headache grew steadily worse, and was most severe at the menstrual period. She was then put under the care of a physician who made the diagnosis of hysteria ; another physician treated her for rheumatism ; a third for chronic meningitis ; a fourth applied electricity, which made her condition worse ; a fifth treated her for rheumatism of the brain. Finally she came under the care of Dr. Hammond, who treated her for brain congestion, together with other symptoms. She had never shown any signs of insanity ; she was always of a lively disposition when not in pain ; she had never had a true spasm. Dr. Hammond placed her in the care of Dr. Dudley, who found that she was well nourished, that her menses were regular, lasting from two to four days, the flow being moderate. She suffered no pain except in the head, but lately had had pain in the side, one week before, and one week after menstruation. She had taken large quantities of medicine, and had been treated by almost every means for relief of her nervous condition. She had been a masturbator for years, but said she had given up the habit some months past. The vulva was so hyperæsthetic that a physical examination was very difficult. The meatus was surrounded by a large caruncle. From a superficial examination he found the uterus retroverted, and the ovaries and tubes correspondingly low. At a second examination, without ether, he was able only to replace the uterus. Saturday last, he examined her under ether ; the left ovary seemed to be small ; the right ovary seemed to be somewhat enlarged, and very sensitive. After consultation, and having obtained the previous consent of the patient, and of her brother, who was a physician, it was decided to make an exploratory operation. The right ovary was found somewhat enlarged, and contained a cyst about the size of an English walnut; the tube was tortuous, and the fimbriated extremity was bound down below the ovary; the ovary and tube were removed. The left ovary was found to be small, and contained a small cyst. The tube was tortuous and bound down to the pelvic floor, and quite close to the intestine; beneath it was a large varix; this ovary and tube, together with the varix, were removed. The patient had since done well.

A point of special interest in the case related to the

VARICOSE VEINS.

This was the third case of the kind which he had operated upon during the past year. In all, there had been failure to relieve the reflex symptoms by various ordinary measures, such as repairing a lacerated cervix, curetting for a fungous condition, the administration of drugs, etc. In the first two cases the pain in the side and other reflex symptoms disappeared after removal of the ovaries and varix. The ovaries were not much enlarged. In the third case, the operation had very recently been performed. Dr. Dudley thought that inasmuch as a varicocele in the male might give rise to almost all kinds of reflex symptoms, so also might an analogous condition exist in the female.

Dr. Putnam-Jacobi thought it would be interesting to make a microscopical examination of the small ovary, and learn whether or not it was the seat of interstitial ovaritis. Two years ago she reported three cases in which the ovaries did not present any very marked lesion when examined macroscopically, but when examined microscopically there were found degeneration of the arteries and the signs of interstitial ovaritis, and she thought this was much more the cause of the severe nervous symptoms which had existed than was any slight cystic degeneration. In fact, a cyst of considerable size might exist and cause no suffering or tenderness when pressure was made over the abdomen. She referred to the case of a young lady who, before coming East, had undergone at least one year's continuous treatment in a Western State for symptoms related, presumably, to an existing enlargement and prolapsus of both ovaries. The treatment by galvanism was continued, and an attempt was made to replace the ovaries, and a boro-glyceride cotton tampon was introduced into the vagina. At first this local treatment seemed to be beneficial. But the relief proved to be only temporary, and the tampon after a time seemed to cause irritation. Local treatment was then discontinued two or three months, during which time the patient received simple massage, to some extent electrical massage, the cold pack with rubbing, and general treatment. Under this plan the pain in the back, and the nervous symptoms generally had entirely disappeared, also the tenderness on pressure over the ovaries, although the organs remained prolapsed and enlarged. Dr. Jacobi had seen at least two or three cases in which the

ENLARGED AND PROLAPSED CONDITION OF THE OVARIES had continued after relief of all the apparently associated symptoms.

Dr. Dudley's specimen was, at his request, referred to the Committee on Microscopy. Referring to the remarks of Dr. Putnam-Jacobi, he expressed the belief that cotton tampons with boro-glyceride produced their beneficial effect by causing contraction of the vessels, and reducing uterine congestion. He did not think that in his case there had been acute ovaritis; the patient had not complained of pain in the region of the ovary until very recently. As to the relief of symptoms by cotton tampons containing glycerine, he had two cases of prolapsus of both ovaries and tubes in which such treatment would cause almost entire disappearance of the symptoms for a month or two, but they would return with each new exposure to cold, overexertion, etc.

Dr. Putnam-Jacobi thought that in the case related by her the uterus was perfectly healthy, and the tampons had produced only slight relief, whereas the general treatment had caused entire cessation of the subjective symptoms.

The President said it was so seldom one found a normal ovary that it was difficult to obtain a sufficient number for demonstration before students. Interstitial ovaritis was very common even in cases in which there had been no history of symptoms associated with it during life.

Dr. W. P. Northrup asked Dr. Dudley whether he had noticed in his cases any of the changes in the features, voice, growth of hair on the face, etc., alleged to take place after removal of the ovaries.

Dr. Dudley replied that he had kept his cases under observation as long as possible, but in none had he noticed a change in the voice, in the growth of the hair on the face, or in any other respect, excepting that usually there was an increase in flesh, and some complained of flushes in the head after removal of the ovaries.

BONY EXCRESCENCES ON THE INNER SURFACE OF THE CRANIUM.

Dr. Thacher presented the skull of a colored woman, about thirty-five years of age, whose clinical history was meagre. She had complained of severe headaches for three years, which she thought were due to menstrual troubles. There were chronic changes in the kidneys, but no diagnosis of kidney-disease had been made by the physician in charge. The urine had, according to the report, been of fairly high specific gravity, and contained no albumin. There was no history of venereal disease. During the past three months the patient had had occasional attacks of vertigo, with loss of consciousness and falling to the ground. The attacks became more and more frequent toward the end of life. She gradually weakened and died. The diagnosis made was epilepsy. There were found at autopsy bony excrescences on the inner surface of the frontal bone, and some calcified masses in the dura mater. The dura was closely adherent to the skull.

REMOVAL OF RIGHT HALF OF LOWER JAW BY SLOW ENUCLEATION.

Dr. Joseph W. Howe sent a patient with the following history : Thomas R——, twenty-three years of age, was admitted to St. Francis Hospital with the following symptoms. On October 4th he received a severe blow on the under surface of the chin, which exposed the bone. The wound was sewed up at St. Luke's Hospital, but failed to unite. The jaw afterward became very much swollen and painful, and he sought admission to St. Francis Hospital. Dr. Howe made an examination in the early part of November, and found an extensive swelling extending from the symphysis to the posterior part of the ramus of the jaw on the right side, and a small hole running up under the chin behind the jaw, which led to the dead bone. The teeth on the affected side were loosened, and the alveolar process stripped bare and rough and bathed in pus. Pressure on the outside, over the jaw, forced a quantity of pus out, both from between the periosteum and bone and external to the periosteum. The periosteum was separated along the upper line of the jaw back to the ramus, and in some parts had been completely destroyed. The lower teeth were removed from the jaw the following day, and a portion of the periosteum separated. This process of separating the periosteum from the bone was repeated at intervals for several weeks, while the parts were thoroughly cleaned and the patient's general health was kept up by tonics and stimulants. With the stripping of the bone, the periosteum gradually thickened so as to increase the swelling on the affected side. Three weeks previous to the first operation he made a careful examination under ether, and separated the greater portion of the periosteum and other tissues from the bone. Pieces of lint steeped in carbolic acid and glycerine were placed on each side of the dead jaw, and the patient was allowed to return to the ward. More swelling and fever followed the operation. On the following Monday he completed the operation, removing the last attachment at the symphysis and pulling out the diseased jaw, leaving behind a good basis for a new jaw in the ossifying periosteum. The slow enucleation of a diseased jaw is a comparatively simple process; very little force and very little cutting are required to remove it. The patient has now a new jaw, which has grown from the old periosteum.

DR. W. P. NORTHRUP presented specimens from a

CASE OF INTUSSUSCEPTION,

with the following history: The patient was a boy aged five months, who was cared for in his illness "between" two or three physicians. The child was well nourished, large of his age, without previous disease. Forty-eight hours before death the only record of him is that he had "severe diarrhœa," with vomiting. During the last twelve hours the vomiting increased in severity, and the diarrhœal discharges became bloody mucus. The patient cried shrilly and had "inward convulsions." The head, rolling constantly from side to side, had worn the hair from the occiput.

On autopsy the abdomen was found tensely distended. The contents of the intestines were yellow, grumous material, with castor-oil, which had recently been administered. The distention was found to be wholly of the small intestine. The stomach was empty, and colon and rectum were collapsed. Lying in the axis of the transverse colon was the tumor shown, which was four inches long, an inch and a quarter thick, round, firm, and of a bluish color, and was found to be an invagination of the cæcum into the transverse colon. The latter was in normal condition; the former deeply congested.

From the hepatic extremity protruded the ileum and mesentery, and a half-inch of the two-inch-long appendix vermiformis. The tissues were deeply congested and dusky. The two innermost layers, lying with their peritoneal surfaces together, were adherent, with tender, recent attachments. On inspecting the splenic extremity, the outermost layer, the transverse colon, was found to be normal. On stripping back the outer layer, the everted and invaginated cæcum was seen. It was like stripping back the prepuce and laying bare the glans.

The color of this packed invagination was almost black; the epithelia macerated and easily swept off, and its surface smeared with tenacious blood stained mucus. The gut was believed to be not gangrenous.

The Society then went into executive session.

Stated Meeting, June 22, 1887.

W. P. NORTHRUP, M.D., VICE-PRESIDENT, IN THE CHAIR.

DR. JOHN C. PETERS presented *portions of intestine* received from Dr. Middleton Goldsmith, of Rutland, Vt., which were interesting only with regard to the history of the case. [The stopper of the bottle had become loosened and allowed the specimens to get dry during transportation.]

Dr. J. H. K——, of Rutland, was taken ill at 9 A.M., June 4th. Dr. Caverly, was called immediately and found him suffering from great pain in the right iliac region. There was no swelling—no tympanites. The bowels had a natural feel.

He described his case as occurring in this wise. He had occasion, just before, to take an enema, which he gave himself. There was no pain during the injection. The first effort to pass anything from his bowels was attended with some pain ; the second with excruciating pain.

At that time Dr. Caverly found him—besides in severe pain—cold to the elbows and knees and pulseless, and his face had the ashen hue of death. He was crying out in severe pain. Dr. Caverly immediately gave him two-thirds grain morphia sulph. hypodermatically, and applied hot-water bags to the extremities, and sent for me.

I found him free from pain, and in answer to my question he said he was in no pain then, and on asking how he had been for several days, he said he had been ailing for some time with indefinite pains through his bowels, with a good deal of pain in the right iliac region for three weeks, and that was all, except that his belly was sore all over.

The first question was, what was the injury? We were not aware *what* it was nor *where* it was. The facts seemed to point to some injury done to the right groin, which looked like a *rupture*, because of the suddenness of its onset, and of the *intestine*, because of the severity of the pain.

We could not help thinking, after witnessing the first shock, that this was a fatal injury—a tear or something of the kind. We did not feel sure, although the symptoms looked that way. We gave opium to narcotism, feeling that that was the only hope of adhesive inflammation and not suppuration. He died at 5.30 A.M., June 8th.

At the post-mortem examination there was found almost universal adhesive inflammation in the right side of the abdominal cavity. There was nothing in the cavity but about a half-teacupful of pure serum. The adhesions were so complete that the parts had to be torn from each other by force. A profile of the torn gut showed the usual long hairy-like attachments. The gut adhered to the liver, and wherever a knuckle of small intestine came in contact with the cæcum or ascending colon it had adhered. After taking out the gut it was filled with water and then pressed with the hand when it was found that it gave way slightly under the grasp as though there was a small rent. The whole length of the gut was examined—the ducts of the liver, the gall-bladder, kidneys, ureters—and they were all intact. There was no injury from the nozzle of the syringe. Bladder and urethra were sound. The duodenum and jejunum were examined ; all were sound.

DR. H. J. BOLDT presented the heart and lungs just taken from the body of a boy eight years of age, which showed

PNEUMONIA AND PERICARDITIS.

that had complicated *traumatic peritonitis*.

On June 12th the patient received a blow upon the belly from the fist of another boy. He went home and went to bed, complaining of intense pain in the abdomen, which began shortly after receiving the injury. His parents, on the next morning, reported that he had had considerable fever during the night and that he complained more or less of pain in the abdomen. On June 13th, Dr. Boldt found him with high temperature, tenderness over the entire abdomen, especially marked in the right iliac region, and he diagnosticated traumatic peritonitis. In the course of two days, however, pneumonia developed, which progressed until the whole of the right lung became involved ; later, the left lung gave evidence of some consolidation. The consultant thought that the right pleural cavity contained fluid, and Dr. Boldt aspirated, but obtained only twenty grammes. During the progress of the pneumonia, the peritonitis was lost sight of. The patient died on the morning of June 22d.

The autopsy revealed diffuse peritonitis, pleuro-pneumonia involving the whole of the right lung, a few ounces of fluid in the left pleural cavity, with a small amount of pulmonary consolidation and marked pericarditis.

Dr. Boldt also presented a specimen of

PYO-SALPINX

on which he operated very soon after rupture of the sac. It was from one of *three* cases in which he had operated soon after rupture. In the first two, peritonitis developed and killed the patients. The third case terminated in *recovery*. The patient came under his observation on June 5, 1887. He had previously seen the patient and made his diagnosis, and intended to operate, but suddenly, on the morning of the date mentioned, she was seized with severe pain in the lower part of her abdomen, accompanied by some vomiting, and she was compelled to remain in bed. There was considerable tenderness, but no other evidence of general peritonitis. On palpation it was also found that the tube, which could be felt distinctly on previous occasions, had collapsed and disappeared substantially. Dr. Boldt operated within two hours, and found that the Fallopian tube had ruptured. There was considerable localized redness about the pelvis, but no peritonitis. The abdominal cavity was thoroughly cleansed by irrigation, and the patient made a good recovery.

Dr. W. P. Northrup presented microscopic sections of

PRIMARY EPITHELIOMA OF THE CONJUNCTIVA,

made from a growth that was removed by operation from the eye of a private patient, a girl three years and two months old, whose parents were living, young and healthy, except that the mother had frequently suffered from obstinate eczema.

The child had a light complexion and clear skin, and has always been healthy, well nourished, and vigorous.

October 14, 1887, she was presented for treatment. There was at the nasal margin of the left cornea a nodule the size of a pin's head, smooth, round, and pale red, and leading to it were three or four enlarged vessels. There were slight lachrymations, photophobia, and diffuse redness of the eye. There was no history of traumatism.

The pathological anatomy was judged to be a simple hyperplasia of gland-tissue at the limbus corneæ, a phlyctænula, which would soon part with its superficial epithelium, empty its contents, and speedily repair. A favorable prognosis was given, the child was seen every day, but at the end of a week no progress had been made toward recovery, the tumor, seemingly, remaining unchanged.

Dr. Agnew then saw the case in consultation, and confirmed both the diagnosis and the prognosis.

In his opinion the hyperplasia, instead of resolving itself, remained as a simple hyperplasia, and would at length return to its normal condition with possible permanent thickening of trifling amount.

In the second week the tumor enlarged somewhat, being perhaps twice as large as when first seen, and at this time it was about four millimetres in diameter ; its base was round, defined, lying partly upon the cornea, partly upon the sclerotic, movable moderately ; its surface was nodular and fissured (cauliflower excrescence), and at its summit there was loss of substance

PRIMARY EPITHELIOMA.

At this time the case was seen by Dr. Agnew and Dr. Webster, who agreed on the diagnosis of epithelioma, and advised prompt removal.

November 19th, Dr. Agnew, assisted by Dr. Webster, removed the growth and cauterized the subjacent tissue.

The conjunctiva was brought together by stitches and the eye closed, having upon it a pad of absorbent cotton lightly held beneath a shield. The patient made a prompt and satisfactory recovery. At this time, seven months after the operation, there remains a smooth cicatrix about four millimetres in horizontal length, and less in width, lying partly upon the cornea and partly over the sclerotic. Upon the cornea it has a pearly opacity visible about three feet distant. The enlarged blood-vessels are no longer seen, and there is no evidence that the scar is not free from any suspicion of returning disease.

The eight microscopic sections shown constituted nearly the whole of the tumor. The microscopic examination of the specimen revealed that it was an epithelioma. This was the opinion of Dr. Delafield, who had carefully studied the sections.

Remarks.—Epithelioma in this locality, whether of the cornea or conjunctiva, is commonly secondary to cancer of the lids. In the case here reported it is primary. In its early stage it is easily mistaken for phlyctænula, though it is attended with less irritation and congestion, lachrymation and photophobia. Its growth is slow at first, then rapidly spreading and invading deeper tissues, and may speedily destroy the organ.

The treatment consists in removing it radically as soon as the diagnosis is made.

The prognosis is bad. The growth usually returns, and speedily.

The present case has passed the seventh month without a symptom of returning, and the prognosis is now considered good.

The Society then went into executive session.

Stated Meeting, September 14, 1887.

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

DR. J. S. THACHER, from the Committee on Microscopy, reported on the specimen presented by Dr. A. P. Dudley on June 8, 1887: The walls of the blood-vessels are thickened and hyaline, as is so often the case in the ovaries of adults. There are also several minute cysts present. No other lesions were found.

DR. FRANK FERGUSON exhibited under the microscope a specimen which showed

THE HOOKLETS OF THE ECHINOCOCCUS,

found in the fluid from a cyst of the liver of a patient who was a resident of Cuba.

Dr. Ferguson also presented a specimen of

TUBERCULOSIS OF THE BRAIN.

The lesion showed itself in large nodules scattered through the substance of the brain. Microscopical examination had revealed tubercle-bacilli in these nodules. There were miliary tubercles in the upper part of the left lung. There was tuberculosis of the bones of the skull and of the scapula, and tubercles were found in other parts of the body. The patient was a child with the history of vaccination a short time previous to the development of tubercular disease, and the question had been raised whether tubercular virus had been introduced by vaccination.

DR. W. P. NORTHRUP regarded the tubercular nodules in the brain as very extraordinary. In only two or three cases had he seen them of such size in the substance of the brain. Less than a year ago he presented a similar specimen to the Society, and regarded it as quite rare.

DR. J. S. THACHER presented a specimen of large

PERI-URETHRAL ABSCESS,

portions of which could be seen in the prostate. There was chronic cystitis, and the walls of the bladder were much hypertrophied. One ureter was very much dilated, its walls were thickened, and the mucous membrane congested; one kidney showed the lesions of suppurative nephritis. There were also pyelitis and dilatation of the pelvis of the same kidney, and in certain parts evidence of diphtheritic inflammation, shown by a patch of yellowish pseudo-membrane so closely adherent that when torn off a raw surface of the underlying tissue remained. All that the patient complained of was a certain amount of pain in the lumbar region, some about the bladder, and incontinence of urine. There was no stricture.

DR. H. J. BOLDT presented a specimen that showed

LOBAR PNEUMONIA—ABSCESS—GANGRENE—TUBERCULOSIS.

The patient was a man who had been taken sick, about six weeks ago, with what was diagnosticated as lobar pneumonia at the apex of the right lung. At the time Dr. Boldt saw the patient the question arose, on account of the profuse fetid expectoration, whether or not abscess was present as a complication. The microscopic examination of the expectoration by Dr. Heitzmann rather confirmed that diagnosis. There was no elevation of temperature above 99° F. The surface of the lung was studded with miliary tubercles, and there was beginning gangrene in the walls of the abscess.

DR. W. P. NORTHRUP presented a specimen sent to him by Dr. Morgan, of Ithaca, N. Y., which was referred to the Committee on Microscopy. It was accompanied by the following history:

CONGENITAL ABSENCE OF THE RIGHT KIDNEY—CARCINOMA OF THE LIVER, COMMON BILE-DUCT, AND HEAD OF THE PANCREAS—CARCINOMATOUS ULCERS OF THE FUNDUS OF THE BLADDER, WITH HÆMATURIA.

The patient was a man, seventy-six years of age, who had been under Dr. E. J. Morgan's observation for several years. Eighteen months before death he began to lose flesh, appetite, and strength. Six months before death he passed from the bladder, once or twice, clear, fluid, bright blood in considerable quantities. The patient felt better after the hemorrhage. Three months before death he again passed blood from the bladder occasionally, the amount varying, and then he seemed prostrated by the loss of blood.

The symptoms which led to diagnosis were progressive emaciation, loss of strength, and hæmaturia. About one month before death, when emaciation was extreme, a tumor, apparently as large as a large orange, was discovered to the right of, and on a level with, the umbilicus. It was firm, ovoid, and movable. The liver was markedly enlarged. There was no jaundice. The countenance was sallow and cachectic. At last there was diarrhœa and occasional vomiting, stupor and transient coma, followed by exhaustion and death.

Autopsy was made, thirty-six hours later, by Dr. Lewis, in presence of Drs. Morgan, Hunter, Turnbull, Baker, and Northrup. The body was extremely emaciated. The liver was enlarged to twice its normal size, its surface was nodular, and the centres of the nodules were softened. The blood-vessels of the surface were dilated and radiating from the centres of the nodules. The common bile-duct was half an inch thick and carcinomatous. The head of the pancreas was involved, and formed the tumor felt through the abdominal wall. The tumor thus formed was three and a half inches long, two and a half inches wide, was freely movable, and of firm consistence. In its centre it was softened, but not diffluent, except just at the junction of the bile-duct with the intestine, where it had found exit into the gut by means of numerous small perforations about the papilla of the bile-duct.

The interesting pathological feature was found in the bladder. The prostate was enlarged, but this was of no pathological significance. The vesical mucous membrane was normal in general appearance at the base and upon the lower half of the anterior wall. Upon the emaining wall there were from fifty to one hundred circular, punched-out ulcers, varying in diameter from five to fifty millimetres. These were clean-cut, free from slough, redder than the mucous membrane surrounding them, and extended through the mucous membrane and half through the somewhat thickened bladder-wall. These ulcers were considered the source of the hæmaturia.

The left kidney was perfectly healthy, was nearly twice the ordinary adult size, and with the exception of three small cysts was normal. The ureter and urethra were also free from lesion. There was congenital absence of the right kidney. A rudimentary ureter could be traced a few inches from the bladder, but there was lost in the tissues.

Dr. Morgon had known this patient as a healthy man for twenty years. There was no history of hereditary predisposition to cancer.

Dr. Northrup referred to cases in which bright fluid blood had been passed in considerable quantities from the bladder without known cause, and the hæmaturia had not recurred.

Dr. Waldstein recalled two cases of hæmaturia, although they had no direct analogy with the case under consideration.

The first patient was an apparently healthy woman, who, without known cause, except there was reason to suppose that she had caught cold, passed a large quantity of blood from the bladder. Without treatment, the patient had no recurrence of the hæmaturia.

On looking up the literature at that time, he found that hæmaturia might be due simply to the effect of cold. As an experiment, he ordered that her feet be placed in ice-cold water, which was done, and she had another attack of hæmaturia.

The other was a case which occurred in the practice of a physician in Berlin. On examination, peculiar bodies were found in the urine, which were presented to Virchow, who declared them to be a parasite which he had described many years before in connection with a similar case, and to which he gave the name *rhabditis*.

DR. WYETH said that

HÆMATURIA

was not an uncommon occurrence in surgical practice. He had reported to the Society, three or four years ago, a case of obscure hemorrhage from the bladder, in which, on careful microscopical examination of the fluid discharged, he found the eggs of the parasite *Bilharzia hæmatobia*. The patient was a young man who went out as a missionary to Africa, where, especially along the Eastern coast, the affection caused by this parasite is very common.

About a year ago he was called to see a woman who, while coming from St. Louis, took cold and had a discharge of blood from the urethra that caused her great alarm.

The third case was one in which he removed the right kidney of a woman thirty-eight years of age. There was recurrent hæmaturia. Dr. Janeway diagnosticated neoplasm as the cause, and the operation proved the diagnosis to be correct. The symptom of pain was very much like that in the case reported by Dr. Northrup.

DR. MARY DIXON JONES presented two specimens of

THE UTERUS REMOVED FOR MALIGNANT DISEASE.

Dr. Jones also presented two specimens of

OVARIES AND TUBES REMOVED BY LAPAROTOMY,

accompanied by microscopic sections.

DR. BOLDT thought Dr. Jones was in error in saying that Dr. Martin considered vaginal hysterectomy a difficult operation. For in any case where the uterus is mobile it is an easy operation, and one completely free from danger.

DR. JONES said that her quotation was taken directly from Dr. Fenger's article on this subject.

The Society then went into executive session.

Stated Meeting, September 28, 1887.

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

DR. MARY DIXON JONES presented

THE OVARIES AND TUBES

removed from a married woman who had had one child and one miscarriage. After the latter she was confined to her bed for five months with probable septic peritonitis. Ever since that time she has suffered from more or less pain in the lower part of the pelvis, which, during the last five months, had been very severe.

The right Fallopian tube was about the size of a hen's egg, was completely closed, all traces of fimbriæ gone, and it was universally adherent, but especially to the posterior part of the uterus, with old and strong adhesions. The right ovary was very thin, and contained bone. There were several parovarian cysts which ruptured during the operation, and there was considerable hemorrhage. The abdominal cavity was washed out with a bichloride solution—1 to 50,000—the peritoneum was united with continuous catgut suture, and the abdominal wound was closed with silk. Reaction did not take place for seven hours. On the second day the drainage-tube was removed. On the third day the temperature was normal, and the patient has since been doing well.

Dr. Jones presented a second specimen of the uterine appendages removed from a woman twenty years of age, who suffered severe pain for six or eight days before each menstrual period, which lasted ten days. Dr. Polk saw the patient in consultation, and recommended removal of the tubes and ovaries, which were evidently very much diseased. Laparotomy was performed, with the assistance of Dr. A. M. Jacobus. Some of the adhesions were so firm and extensive that it became necessary to ligate them, and then divide them with the knife. Both pedicles were very large; they were secured with ligatures, and cauterized. Both tubes were closed. The drainage-tube was employed. This patient was put to bed, and her symptoms disappeared, and yet at the operation the extensive disease was found as shown in the specimens, which were liable to rupture at any time, and give rise to serious complication, if not fatal result. She did not approve of attempting to support the appendages by means of a pessary, neither had she seen a case in which cerebral congestion had been produced by suddenly stopping menstruation.

Dr. Carpenter asked Dr. Jones how many cases she had seen in which pyo-salpinx ruptured previous to operation.

Dr. Jones had seen *one* that was proven by post-mortem.

Dr. Boldt had met with *six* cases.

Dr. A. P. Dudley had encountered *one* case.

Dr. Ferguson had made a post-mortem examination in *one* case.

Dr. H. J. Boldt exhibited

AN INTUBATION-TUBE THAT HAD AROUND ITS HEAD AN INCRUSTATION OF CALCAREOUS MATERIAL.

He thought it possible that, in some cases, ulceration might be produced by such a deposit around the upper part of the tube. The case in which it was used was an apparently hopeless one, but the child made a fairly good recovery.

Dr. Wyeth mentioned recent statistics, published by Dr. Stone in the *Pittsburg Medical Review*, of thirty-two cases of intubation, with twenty-one deaths, and the writer concluded by condemning the method.

Dr. W. P. Northrup had seen a tube that had been in the œsophagus fifty-two hours, and was covered entirely with a deposit similar to that seen upon Dr. Boldt's tube. In that instance it was accounted for by the fact that the bichloride of mercury had been used in the treatment of the case. He had never seen an ulcer at the head of the tube that did any harm. The ulcer which did harm was at the lower end of the tube, and upon the anterior wall of the trachea.

The tube was referred to the Committee on Microscopy for examination and report as to erosion of its surface and precipitation of lime salts, lime-water having been used in the treatment of the patient.

Dr. A. P. Dudley presented a specimen of

MALIGNANT (?) DISEASE OF THE OVARY,

with the following history: Annie D——, single, thirty-four years of age, and always healthy; menses regular, and lasting one week, until last February, when the menses began to appear every two weeks and remain six days. She never had any pain with the menses or otherwise until March, when she was troubled with gravel, and micturition was attended with great pain and the passage of small quantities of urine. The gravel disappeared about the middle of April. She then began to have pain in the right inguinal region, which continued to be very severe, and lasted until about the first of August, when it changed to the character of a weight in the abdomen. The menses continued to appear twice monthly until the 6th or 7th of July, when they lasted only two days. In August there was only a show. She first noticed "a bunch" in the right side about the first of April. It increased in size very rapidly, and was attended by the pain, as previously mentioned; she lost flesh rapidly during the past three months, and when seen by me, August 25th, she presented the typical *facies ovariana.* Examination at that time revealed a large tumor in the abdomen, giving to the touch the impression of a multilocular ovarian cyst with thick walls and contents. It was quite movable from side to side, but not from above downward—the greatest portion of the tumor being to the left of the median line of the abdomen, and the upper portion seemed quite hard.

A DISTINCT PERCUSSION WAVE COULD BE OBTAINED.

Digital examination showed the hymen intact, vagina normal, and womb pressed forward against the pubes. No sound was introduced into the organ. The left cul-de-sac was occupied by a hard portion of the growth. No bimanual percussion could be obtained considering the duration of the growth and its size. He anticipated finding *malignant* disease, and so stated to her physician; operation advised as early as possible. She entered St. Elizabeth's Hospital at once, and the operation was made on August 31, 1887. Incision three inches long, through linea alba. The peritoneum was found thickened to one-eighth inch anteriorly. The whole anterior surface of the growth was adherent to the anterior abdominal wall. This was stripped off; no ascitic fluid escaped. When tapped, the growth proved to be multilocular, with the cysts containing widely differing contents—some jelly, some clear limpid fluid, some a cheesy matter, and some blood. Great care was taken not to allow any of the contents of the tumor to enter the general peritoneal cavity. The growth had a long pedicle, which was easily ligated. The right ovary was found to be the size of a small orange, cystic and adherent, with corresponding tube some enlarged; both were removed. She rallied quickly from the shock, aided by rectal injections of brandy and water. There was nothing worthy of mention concerning the after-treatment, except that the temperature never went above 101½° F. (next day), and the pulse never above 107. She left the hospital on Monday, September 26, 1887, cured.

Dr. Dudley remarked that the first case in which he operated was similar to the one just reported. The patient suffered so severely from shock that he was obliged to turn her upon her back, when some of the contents of the tumor entered the peritoneal cavity. At the time of the operation the pelvis was apparently perfectly healthy, but in five months the woman died of return of the sarcoma in the pelvis.

The specimen was referred to the Committee on Microscopy.

Dr. F. C. Fuller mentioned a case in which there had been a notable duration of life with malignant disease, there being a history of active recurrent cancerous disease of the pelvic organs from December last until the present time.

Dr. Dudley also presented

THE SAC OF AN OVARIAN TUMOR

which, together with the contents, weighed seventy pounds. The sac weighed eight and a half pounds. The tumor was removed from a woman fifty years of age, who was married when twenty-seven years old, and had had substantially a normal history of menstruation. Her labors had been normal, and she had not had a miscarriage. Her last pregnancy occurred sixteen years ago. She had never suffered from irritation of the bowels nor vesical symptoms. The chief, and nearly the only, symptom was constant pain, varying in severity. Her body, at the time of the operation, was lighter than the tumor, and he operated without the expectation that she would recover from the shock.

From the pubis to the umbilicus the distance measured 15 inches; from the anterior superior spinous process of the ilium to the umbilicus, 18½ inches; from the ensiform cartilage to the pubis, 30 inches; half the distance around the body, 26½ inches. The tumor was universally adherent. The woman lived five days, and died from complications.

Dr. Dudley further presented

THE OVARIES AND TUBES

removed from a woman thirty-four years of age, who was married at the age of fourteen, and had had two labors and three abortions. Her general health had been very poor, and locomotion had been difficult. There had been periodical gushes of water from the vagina. Hydro-salpinx on the left side was diagnosticated. She had gonorrhœa several years ago. The leucorrhœal discharge which she had, had never been purulent. The uterus was retroverted and bound to the pelvis, and there were extensive adhesions of the ovaries and the tubes on both sides. The operation confirmed the diagnosis, and the case was reported to illustrate how close the diagnosis might be in a case that was carefully studied.

He mentioned a case reported to the Maine Medical Association, in which the diagnosis of hydro-salpinx was based largely on the periodical occurrence of copious watery discharges from the vagina. Removal of the tubes relieved the patient entirely.

Dr. Ferguson remarked that in thirty out of sixty or seventy-five post-mortem examinations he had made in cases of salpingitis the tubes were found closed.

Dr. J. S. Thacher, by the kindness of Dr. Jacobus, was able to present an extreme case of

CONGENITAL UMBILICAL HERNIA, WITH MARKED ABSENCE OF DEVELOPMENT OF THE ANTERIOR ABDOMINAL WALL.

It was interesting on account of the size of the mass, and the obstacle which it offered to delivery.

The child was a full-term child, and its movements were said by the mother to be active till a few hours before labor-pains began.

The mother was a primipara, twenty-four years of age, and in good health. She had had two slight falls, a few weeks before, but with no apparent injury. There was no history of congenital malformations in the families of either parent.

The membranes ruptured fourteen hours before pains began, and the waters contained some fæcal matters, as they were discharged at intervals through the day. When examined, soon after the labor began, a transverse presentation was found, and protruding from the uterus, besides an arm, a peculiar mass, feeling like a portion of the amniotic sac, containing about a pint of fluid, and something like a placenta. It was found to be attached by a pedicle high up.

Podalic version was performed, and the child extracted. During version the bag ruptured. The intestines were born with the hips. The liver, also outside of the body, interfered some with the birth of the shoulders. The cord and placenta were normal.

On examination after birth, there was found an absence of a considerable portion of the anterior abdominal wall, about four inches in extent, the edges of which were continuous with the thin membrane which formed the sac mentioned. In this, and quite outside of the trunk, lay the liver, spleen, left kidney, and most of the intestines, while the stomach and uterus protruded partially. The heart was displaced downward, but inside the trunk. There was marked right lateral bending of the spine.

Dr. F. C. Fuller presented a

DERMOID CYST OF THE OVARY,

removed from a patient thirty-seven years of age, married, who came to him, last June, complaining of irritation of the bladder, which he regarded as due to mechanical pressure. There was occlusion of the vagina, which had existed during married life. There was a tumor in the right side of the pelvis, which pressed the uterus forward and to the left. He tapped it with a small needle, and obtained, thick, yellow pus. He then aspirated the sac and drew off one pint of pus, and the patient remained relieved for some time. She got into the care of another practitioner, but finally came under Dr. Fuller's observation again, when the evidence of a pelvic abscess was obtained, which he opened into the vagina. Soon after, the symptoms led to the diagnosis of rupture of the abscess into the peritoneal cavity, and laparotomy was performed immediately. It was with very great difficulty that he succeeded in removing the pelvic contents and inserting a drainage-tube through the vagina. The patient died of exhaustion. The diagnosis of dermoid cyst was made by exclusion, and confirmed by the few hairs which protruded into the vagina when the abscess was opened.

Dr. Fuller also presented a specimen of

INTRA-CAPSULAR FRACTURE OF THE NECK OF THE FEMUR.

The patient was a woman eighty years of age, and in excellent health. She died four days after the accident, of cerebral apoplexy. The autopsy showed that there was no impaction, which was thought to be present at the time the diagnosis of intra-capsular fracture was made.

Dr. Dudley assisted Dr. Fuller in his ovarian case, and he remarked that he once had a similar one in which there was a pelvic abscess separate from the dermoid cyst. These were the only cases which he had seen where there was a simultaneous development of an ovarian tumor and a pelvic abscess.

Dr. Frank Ferguson presented a specimen of

ANEURISM OF THE THORACIC AORTA,

which was interesting chiefly on account of its size. It was removed from the body of a man fifty years of age, a native of Italy, single, an artist by profession, who was admitted into New York Hospital, into the service of Dr. George L. Peabody, on September 22, 1887. He gave no family history bearing on his case. He used alcoholic beverages moderately. Fifteen years previous to admission he had had a chancre, which was followed by an eruption. Three years ago he began to suffer from pain in the left shoulder, which gradually grew worse ; a year later he began to cough, with whitish expectoration. Ten days previous to admission he began to suffer from shortness of breath, to grow hoarse, and to suffer from dysphagia. His appetite was poor ; he was constipated, but his pain was his chief complaint.

On admission his temperature was 99° F.; respiration, 22 ; pulse, 84. He was poorly nourished ; and on physical examination, all over the chest anteriorly, except at the upper part of the sternum and at the apices, the percussion-note was hyper-resonant, and over this area were heard a great many sonorous and sibilant râles. The cardiac sounds were faint, with the second aortic sound slightly accentuated. The pulse was fair in tension ; the peripheral arteries were tortuous and atheromatous. There was a slight arcus senilis. In the interscapular region, to the left of the median line, there was a bulging of the thoracic wall, measuring vertically 12 inches and transversely 8 inches. Over the entire tumor was expansile pulsation and flatness, but no murmurs could be heard.

Examination of urine.—Yellowish in color ; reaction alkaline ; sp. gr. 1.010 ; albumen present in small amount. On microscopic examination, granular casts and triple phosphates were found.

While in the hospital he suffered considerable pain, which was easily controlled by morphine, and he was fairly comfortable till ten minutes before death, when he began to cough up blood and suffer intensely from dyspnœa. He died three days after admission.

Autopsy.—The left lung was bound to the thoracic wall by old adhesions, and it was also firmly connected with a large aneurismal sac, which occupied the posterior half of the left pleural cavity, above the seventh rib. This sac measured transversely 22 ctm., or 9 inches, and vertically 19 ctm., or 7½ inches. It extended 10 ctm. to the right of the median line. The third, fourth, fifth, sixth, and seventh ribs were eroded, the third and seventh partially, and the rest completely. The dorsal vertebræ corresponding to these ribs were also eroded, the fourth, fifth, and sixth most extensively. The sac communicated with the aorta by a circular opening 3 ctm. in diameter, and 4.5 ctm. below the origin of the left subclavian artery. This sac was filled with laminated fibrine and recently coagulated blood. The œsophagus and trachea were pushed forward, and to the right. The œsophagus, laid open, appeared normal. In the median line of the posterior wall of the trachea, on a level with the division into the primary bronchi, was an opening half a centimetre in diameter. Surrounding this opening the wall of the trachea was much thinner than normal, and it tapered to the thickness of tissue-paper at the edges of the opening. One centimetre from the opening two of the cartilages of the trachea were exposed, to a limited extent, through the mucous membrane of the tube. The heart was normal in size ; its valves were competent, but the muscular tissue was pale. The aorta throughout was atheromatous.

Dr. Ferguson also presented a specimen of

CARCINOMA OF THE STOMACH, UNATTENDED BY GASTRIC SYMPTOMS.

A negro, aged fifty-seven, a native of the United States, married, and a caterer by occupation, was admitted into New York Hospital, into the service of Dr. G. L. Peabody, on August 30, 1887. His mother died of consumption, and he also lost a son from the same disease. He denied rheumatic and venereal trouble. He had a moderate alcoholic habit. Thirty years ago he had had malarial fever. For a period of one year, commencing eight years ago, he had had frequent pulmonary hemorrhages. For five years he had had a cough, with muco-purulent and bloody expectoration, night-sweats, afternoon fever, and he became weak and emaciated. At the expiration of five years he partially recovered his health. He had coughed up no blood for the past eight months. Eighteen months ago his feet began to swell, and he passed his urine more frequently and in larger quantities, its color being pale. He had muscæ volitantes, tinnitus aurium, and considerable headache, limited to the left side. His eyesight was impaired, and he had become short of breath, especially on exertion. Recently he had had no further pulmonary symptoms. His appetite was fair. His bowels were irregular, but he gave no other symptom referable to the digestive tract. His chief complaint was pain in his back. On admission his temperature was 97° F.; respiration, 24 ; pulse, 74.

Physical examination.—All over the chest, anteriorly, percussion was hyper-resonant, and expiration prolonged. Posteriorly there was flatness over both bases, with diminished voice and breathing. The heart was apparently enlarged ; the first sound was markedly accentuated at the apex. The liver-area was diminished in size. The spleen-area was normal. The tension of the pulse was rather high. There was marked œdema of both legs.

Examination of urine.—Yellow ; acid ; sp. gr. 1.012 ; albumen a trace ; granular casts and blood-cells found under the microscope.

Shortly after admission he became delirious, and the œdema increased. His urine was passed involuntarily; it contained granular and hyaline casts. He became comatose later, and died on September 27th. His temperature was throughout subnormal.

Autopsy.—The heart was small, and its muscular tissue brownish. The valves were normal in capacity and apparently competent. The aorta was atheromatous. There was emphysema of the right lung along its free border; in other localities it was œdematous. The left lung was intensely œdematous. The kidneys showed the lesions of chronic diffuse nephritis, and contained a great deal of fat. The stomach contained six or seven nodules, varying in size from half an inch to one inch or more in diameter. One was distinctly pedunculated, and some of them were slightly umbilicated. Microscopical examination revealed that they were carcinoma. This disease had not been suspected during life.

DR. BOLDT referred to a case in which the patient died of consumption. The only symptom referable to the stomach during life was some pain after eating, but the autopsy revealed carcinoma of that organ.

The Society then went into executive session.

Stated Meeting, October 12, 1887.

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

DR. A. P. DUDLEY presented the

TUBES AND OVARIES

removed from a woman fifty-one years of age. They did not show any pathological change. The history of the case contained the principal point of interest. She was married when thirty-three years of age, was the mother of four children, and had never had a miscarriage. Her first delivery was normal and not rapid. In her last labor, eleven years ago, she was delivered rapidly of a child that weighed ten and a half pounds, and had never been well since. During these eleven years she had been in bed most of the time ; at no time had she been able to walk the distance of the length of a block. During the last three years she had not been out of bed, except to use the commode. Her menstrual life was about normal, and she never had a leucorrhœal discharge. There was chronic constipation, but no vesical symptoms.

Her chief complaint was concerning

PAIN IN THE SPLENIC REGION,

which was not constant, but was severe whenever she was upon her feet. There was fixed retroversion of the uterus. A great variety of diagnoses had been made, and among these aneurism. Dr. Dudley was unable to obtain any positive evidence of aneurism, although there was marked pulsation directly over the abdominal aorta, with a moderate amount of thickening, apparently, just below the umbilicus. He recommended exploratory incision, and performed the operation, October 11th. He introduced the whole hand into the peritoneal cavity, found the spleen small and without adhesions. Nothing pathological was found at the site of the pain of which she had complained. The aorta was normal, except that it apparently bifurcated high up, and at that point it was quite large.

THE MENOPAUSE WAS COMPLETE ONE YEAR AGO.

In the pelvis the lower part of the descending colon was adherent to the broad ligament and ovary. The uterus was retroverted and firmly adherent. The uterus was not reduced in size.

Adhesions were broken up and the uterus liberated. Both broad ligaments were filled with knotted veins. Both tubes and ovaries were removed. Sufficient of the broad ligaments was caught up to include the varices and the round ligaments, and when this had been done the uterus assumed the normal position, and was thus also separated from the fresh surface of the adhesions that had been broken up behind it. The patient was doing well, had not needed any opium, and had been free from pain. The question arose, *How much good can be done by removal of the tubes and ovaries after the menopause?*

ECHINOCOCCUS-CYSTS OF THE LIVER AND PERITONEUM.

DR. IRA J. VANGIESON, presented specimens which were taken from the body of a middle-aged German woman, who was delirious a few days before death, but gave no symptoms referable to the parasitic lesion. The cysts occurred in the following situations:

(1) Partially embedded in the right lobe of the liver, a little external of the gall-bladder, there was a soft globular sac about two and a half inches in diameter. The wall of the sac contained a few blood-vessels, and the exposed surface of the sac was slightly adherent to the surrounding tissues. (2) Attached to the omentum in the right lumbar region was an oval sac 3½ by 1½ inches in diameter. (3) In the hypogastric area, adherent to the omentum, was a kidney-shaped sac, 4 by 1½ inches in diameter. (4) In the left inguinal region were two sacs; one, oval in shape (2½ by 1 inches in diameter), lying against the abdominal wall; the other, somewhat smaller, lying loosely over the sigmoid flexure. (5) The right inguinal region contained a group of four smaller sacs, lying distinct from each other on the fascia of the psoas-muscle. One of this group was adherent to the vermiform appendix near its junction with the cæcum. (6) Embedded in the mesentery, about the size and shape of the mesenteric glands, were several other sacs. (7) On the anterior surface of the uterus were a number of small cysts about the size of peas; on the anterior lip of the cervix uteri was a larger one, about 1½ inch in diameter.

These sacs, when opened, were found packed with larger and smaller translucent, mottled gray and white cysts, associated with a varying amount of soft, white material. The cysts were collapsed and tightly rolled, so as to become spindle-shaped. Frequently, several cysts were rolled together in a single coil. Scrapings from the cyst-walls, examined microscopically, showed fat-globules, granular matter, cholesterin crystals, and fragments of the peculiarly laminated outer wall of the cysts. Hooklets and scolices were not found, so the cysts might be termed sterile.

THE PRESIDENT regarded the case as interesting, because of the rarity of the peritoneal echinococcus-cysts in such numbers. He could recall only two cases where the peritoneal cysts were so numerous and large.

Dr. VANGIESON also presented a specimen of

PENDULOUS UTERINE MYOMA,

which he found suspended from the cervix uteri and completely occluding the vagina. It was taken from the body of an old woman who died of acute exudative peritonitis in Charity Hospital. The tumor closely resembled a human heart, measured 4½ by 5 inches in diameter, and weighed a little over one pound. Its long axis corresponded to the long axis of the vagina. Its base was against the cervix, to the posterior lip of which it was attached by a slender flattened pedicle half an inch broad and one-eighth of an inch thick. The discolored apex protruded from the vulva about one and a half inch. The tumor was moderately adherent to the posterior wall of the vagina.

DR. JOHN C. PETERS, Chairman of the Committee appointed to prepare a

MEMORIAL NOTE ON THE DEATH OF ALONZO CLARK, M.D., LL.D.,

read the following, which was accepted and ordered to be published in the minutes:

It is eminently proper that the New York Pathological Society should honor the memory of Dr. Alonzo Clark, for he was always its friend, and often its teacher and counsellor, for nearly fifty years.

But we are not called to mourn his loss as those who cannot and will not be comforted, for he lived an unusually long, healthy, honored, and useful life, which was only ended by great age and its attendant infirmities, and these, moreover, were so wearing that he longed to be released from them. Even under these distressing circumstances, we cannot go as far as Diedrich Knickerbocker, who says the death of a great man is no very inconsolable matter of sorrow, and that no fact is more manifest than that the death, even of a very wise man, is a thing of very little importance: nor agree with Pliny, who says: "Of what consequence is it that individuals come and go? Sage follows on the footsteps of sage; one hero steps out of his triumphal car to make way for another, and of the proudest monarch it is merely said, 'He slept with his fathers and his successor reigned in his stead.' The world cares but little for their loss, and if left to itself would soon forget to grieve. It is the biographer and the historian who generally have the whole burthen of grief to sustain. They inflate the people with sighs they never heaved, and deluge them with tears they never dreamed of shedding." All this is not true of Dr. Clark, who has, and long will have, many sincere mourners.

It is more than ten years ago since many of us noticed, with sincere regret, that age and increasing weakness no longer permitted him to attend our meetings with his usual regularity, and that meant a great deal for Dr. Clark, as it is said that before this, in his long career, he had never missed a college, hospital, or consultation appointment. Many months ago we were pained to find that progressing debility rendered intercourse with him uncertain and difficult; still he was always pleased to see his old friends, and it was frequently evident that he had not lost his interest in this Society.

Finally he could only sit, with mingled Christian and Socratic resignation, calmly noting the slow but relentless approaches of that Great Destroyer which he had so often baffled in the service of others, and so frequently triumphed over.

But before we were called upon to pay him our last tributes of love and respect, we were suddenly summoned to mourn the loss of his great and pleasant rival, Dr. Austin Flint.

Dr. Clark was over eighty-one, and had been a leader in this Society for forty-three years. Dr. Flint was only seventy-four years of age, and had worked with us usefully and agreeably for twenty-seven years. But Dr. Flint was still healthy, active, and elastic in body and mind, and altogether excellent. Some of us expected to meet him for a decade or more. Like Dr. Clark he was loaded down with professional honors, and had a very large clientage of friends and doctors. He, too, was a great medical teacher and a famed hospital and consulting physician. Dr. Clark had often wished to reach his eightieth year, and hoped to do this with his eyes undimmed and his strength not greatly abated. Dr. Flint had frequently signified his preference for sudden death. The wishes of both were granted. After an unusually busy, honorable, and successful day, Dr. Flint was stricken down so quickly that he died and made no sign ; and so mercifully that we had the firm assurance that he felt no suffering. Dr. Clark was still looking quietly toward that eternity for which he had no fears, and submitting to his lingering fate with dignity and complete composure. If Dr. Flint had lived another short year he would have been publicly crowned in Europe and America with the highest medical honors which had ever been given to any American physician; and Dr. Clark had long been worthy of them. But, as it were, in the twinkling of an eye one great New York consulting physician was dead and another was completely incapacitated.

"Both had minds of singular breadth and wisdom, accustomed to rest their judgments upon an enormous range of experience and learning; to hearts as large and warm as their intellects were comprehensive they added a gracious gentleness and dignity of nature which adorned and rounded out characters of exceeding completeness and loveliness" (C. A. DANA).

Both had often witnessed every shade of human suffering and agony; both had watched death in all the numberless and dreadful varieties in which it appears to great medical counsellors and hospital physicians, in widespread epidemics, malignant fevers, and swift inflammations, and in all the slower but equally relentless steps of malevolent chronic disease. The one was spared all warning of his end, the other was destined to drain the last bitter dregs of the cup of misery. But so completely master of himself was Dr. Clark, that we saw no signs of unmanly fear, or even of impatience; and so completely had he always kept fully abreast of his profession that he was not called upon to sorrow for any great professional mistakes or deficiencies, much less of crimes. His medical conscience was clear, and his end was that of the good and faithful servant who had increased his talents seventy and even an hundred fold.

We should remember both Drs. Clark and Flint because much of their best and most original work was done with and for this Society, and that in such abundance that the mere record of it fills several hundred pages of our archives.

It is believed that Dr. Clark favored this Society more than any other. He joined it in the first year of its existence, viz., in 1844, and much of his newest and most interesting researches were first made public at our meetings. He from the very first reached a very prominent position in our ranks, for he was then over forty years of age, and in the very prime of his health and fame. He was already an excellent medical scholar; a singularly good clinical and didactic teacher; a noted hospital physician; a favorite counsellor of scores of general practitioners; and had long been doing earnest and honest work in the rapidly spreading fields of pathological science.

It is believed that Dr. Clark then and always regarded this Society as the most useful medical organization in this city. He certainly strove very hard to make it so. Your Committee has gone over much of our earlier records, and have been surprised at the very large number of rare and important specimens which he exhibited years ago, for himself and others; for many physicians preferred the clear and scientific way in which he could bring out their peculiarities to their own more imperfect efforts. He was then, as always, a most industrious and painstaking man, and it is scarcely an exaggeration to say that sooner or later he exhibited specimens of almost every fatal disease to which the human frame is liable.

Dr. Clark was gifted by nature with a most commanding, dignified, and comely presence. He was so large that it often seemed as if he must be unwieldy; but he was so careful and methodical in all his ways that he ever appeared untiring. He was not alone in some of these characteristics, for there were giants in those days in the New York Pathological Society, and the magnificent forms of Willard Parker, Valentine Mott, William H. Van Buren, C. R. Gilman, Middleton Goldsmith, George Wilkes, T. M. Markoe, G. A. Sabine, L. A. Sayre, J. T. Metcalfe, S. S. Purple, T. M. Halstead, and others were often seen among us. But Dr. Clark towered above them all, the largest and most symmetrical, often the most learned and earnest, yet frequently the most genial and winning; for his kindliness and humor were quite as marked as his more solid qualities. The size and shapeliness of his person were only rivalled by the breadth, depth, and pleasantness of his mental and moral characteristics.

These physical and mental giants were closely matched in intellect and knowledge by many smaller personages which so often inclose precious gifts, like John A. Forrey, John A. Swett, Edward Delafield, James R. Wood, William C. Roberts, C. E. Isaacs, Robert Watts, the two little Peterses, and many others; yet Dr. Clark thought and spoke with ease and confidence among the quicker, and perhaps brighter, sallies of his less ponderous colleagues. He sometimes appeared very deliberate, for one of his most marked characteristics was his extreme accuracy. His love of precision led him to as careful a study with the microscope as the instruments of that time would permit, and it is believed that he was not only the earliest medical worker with the microscope in this city, but the first American physician to own a really good apparatus. However this may be, he certainly made almost all the earliest microscopical examinations which were made in this Society, for the specimens of others were constantly referred to him for more minute study. He continued to do this work with few rivals until the microscope fell into the hands of specialists; but the lamp which lighted up his own magnifier never went out until he had completely retired from practice.

In those early days the doctor's watch took the place of the clinical thermometer, and not only measured time, but tried to measure fever. Dr. Clark's timepiece was more accurate than the microscopes and thermometers of those times. He had many appointments to keep each day at college, hospitals, and especially at numerous consultations. Although he never kept a private carriage or used a hired one, he was never behind time. He never was seen hurried, much less excited, nor did he ever appear fatigued. He was always true to time, and as cool. He seemed as incapable of forgetting the exact hour of an appointment as he was of deliberately violating a promise. His high sense of honor and honesty he carried into all the affairs of life, especially into the study and treatment of disease. He knew when to give, and especially when to withhold, his medicines. He had so often seen nature do much that he had learned when to trust solely to her; but in peritonitis he frequently gave, and that successfully, hundreds of grains of opium in a day, for days and almost weeks together.

He went about on foot, because he regarded a certain amount of exercise necessary for his bulky frame. Besides, he was opposed to all display and ostentation. He was obliged to keep a score or less of horses at his country-place at Darien, Conn., but he used no carriages or horses in this city. He unobtrusively went his daily rounds, as if he was one of the least important of doctors. His love of truth and dislike of display also led him to prefer his ears to any instruments in examinations of the chest, although he and Dr. Cammann discovered and perfected the stethoscope which has been the favorite one all over the world for thirty years or more.

Our archives also preserve many instances of the remarkably earnest, honest, and masterful manner in which Dr. Clark led or followed the numerous discussions which arose in this Society. It was always evident that he had no fixed opinions or beliefs except such as were deeply founded upon well-assured facts. He often stripped off the rosy glamour with which more enthusiastic and imaginative men tricked out mere notions or hypotheses; but it was always evident that he had applied the same wholesome pruning process as relentlessly to his own visionary ideas, if he ever had any. He was always very courteous and fair in debate; he mingled great gentleness with extreme firmness, and was rarely worsted in argument for the lack of assured facts or solid reasoning. He was heard at his best in old-time discussions with Drs. Parker, Van Buren, Markoe, Isaacs, Metcalfe, Roberts, Watts, Peaslee, Dalton, Post, Jacobi, Buck, Sands, Krackowizer, Sabine, Shrady, and others. It was only occasionally that we could see how sharp and heavy in retort he could be. His bearing in these discussions was perfect. He never showed the slightest sign of impatience or annoyance, and used only the simple and clear eloquence of great and accurate knowledge, occasionally lightened up by a little humor, in preference to downright contradiction. He always appeared like one who had earnestly striven, and successfully, to learn all that was positively true in medicine, and had equally carefully avoided all that was immature and uncertain. Occasionally, if some new-comer endeavored to show that he knew more than the whole Pathological Society, Dr. Clark would bonnet him relentlessly.

Fortunately the Pathological Society has always had such reliable leaders, commencing with our first President, Dr. John A. Swett. The number of those who strove for mere verbal or windy victories has always been small.

The New York Pathological Society, although founded by young men, all under the thirties, is the oldest association of the kind in the world, preceding the London Pathological Society by two years. But Dr. Clark quickly joined it, as did Parker, Van Buren, and others of like stamp. Dr. Clark was long one of our great leaders. At the time of our founding he was the foremost pathologist in this country, and by his hard work and enthusiasm gave much impetus to the new Society and to the study of pathology all over the land. As far as mere pathology is concerned, the places of Drs. Clark and Flint have long been filled, but as wise, considerate, experienced, and kind physicians, advisers, and friends, their loss will long be felt by those who knew them best.

Dr. Flint will live in his writings. The public fame of Dr. Clark is especially intrusted to the College of Physicians and Surgeons, to this and other societies, and to the hospitals he served so long and so well. They will not let the words of Diedrich Knickerbocker become true and his name forgotten.

J. C. PETERS,
W. M. CARPENTER,
W. P. NORTHRUP,
Committee.

Stated Meeting, October 26, 1887.

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

DR. G. C. FREEBORN, from the Committee on Microscopy, reported on

THE INCRUSTED INTUBATION-TUBE

presented at the last meeting by Dr. H. J. Boldt.

"The incrustation proves, on micro-chemical examination, to be calcium carbonate mixed with a large percentage of organic matter. The polish of the gold-plating is perfect except in the immediate vicinity of the deposit. Around the spots of incrustation, for a small fraction of an inch, it is roughened, and appears as if it had been acted upon by some corrosive agent. In no place uncovered by the incrustation was the plating wanting, though in a few spots it appeared, on examination with a magnifying-glass, as if it had been scraped with a rough instrument."

[At the meeting held on November 9, 1887, Dr. Freeborn made the following supplementary report :]

"On removing the deposit of lime salt, by means of hydric chloride, the gold-plating underneath was found, upon examination with the microscope, to be perfect, and the appearance of corrosion, described above, was probably due to a thin deposit of lime salt.

"Experiments were also made with solutions of mercuric chloride, varying in strength from 1–2,000 to 1½ per cent. These solutions were applied to the gold-plating of the tube, and allowed to remain in contact with it from ten to thirty minutes. The tube was then washed in distilled water, and upon microscopical examination no erosion or change in the character of the gold-plating could be detected."

Dr. Freeborn further reported on the specimen of

"MULTIPLE ULCERS" OF THE BLADDER,

presented by Dr. Northrup, that, on microscopical examination, they were found to be *diverticula* of the bladder.

DR. W. P. NORTHRUP made the following supplementary report on the specimen presented by him on September 14, 1887 :

"The numerous small, round, punched-out excavations, having a deep-red base and wall, had been found, on microscopic examination, to be not ulcers, but diverticula. The discoloration was post-mortem. On section, the mucous membrane of the bladder was found to wind around the sharp edge of the excavation, follow the wall of the undermining cavity and traverse the base and opposite wall, and become again continuous with the mucous membrane of the bladder.

"The following was the best explanation they had been able to find of this condition: Whatever obstructs the flow of urine, such as stricture or enlarged prostate, tends to hypertrophy of the bladder-wall. 'The muscular coat is thickened uniformly, or assumes a trabeculated appearance.'[1] As to the diverticula, 'they are produced by a protrusion of the mucous membrane between hypertrophied bundles of muscle-fibres.'

"Pressure has forced the mucous membrane out between these hypertrophied bands, forming pouches or herniæ of mucous membrane.

"As to the *hæmaturia*, the only explanation they could offer was that of diapedesis. 'Without recognizable changes in the walls of the vessels, all the elements of the blood might become extravasated by passing without rupture through the walls of the vessels,'[2] under certain conditions. There was no post-mortem evidence of any rupture of vessels nor ulceration, now that the diverticula were explained."

Dr. W. M. Carpenter remarked that it was a recognized principle in surgery *not* to empty the bladder wholly at once in cases of retention of urine of several hours' duration, because of the liability to the occurrence of hemorrhage by diapedesis.

Dr. W. P. Northrup presented, in behalf of a candidate, a specimen of

CARIES OF THE SPINE.

Dr. A. P. Dudley presented a specimen of

MATERIAL REMOVED FROM THE CAVITY OF THE UTERUS,

with the following history: The woman was sixty-five years of age, and the mother of four children. All her labors were normal. She passed the menopause thirteen years ago without accident. About three years ago she lost her husband suddenly, and she did not recover from the shock for a long time. About two years ago she began to have uterine hemorrhage, which had continued more or less constant—slight flow for a few days, and recurring after a short interval—until last July, when it became profuse. She brought with her the diagnosis of uterine fibroid, but Dr. Dudley was unable to obtain any evidence of that affection. The uterine cavity was only three inches deep; the os and cervix showed no lesion. There was no evidence of peri-uterine disease. There had been no odor to the blood at any time. There had been but little pain, except pain in the back. There had been no loss of flesh, and there was no cachexia.

---

[1] Delafield and Prudden: Handbook of Pathological Anatomy and Histology.
[2] Ibid.

Dr. Dudley dilated the uterus rapidly and removed the mass presented, which

COVERED THE ENTIRE INTERNAL SURFACE OF THE ORGAN.

Dr. H. C. Coe had examined a portion microscopically, and reported that it had the appearance of fungosities; it might be a myxomatous polypus that had undergone degeneration, and it was possible that it might be an adenomatous growth; but he was unable to find any evidence of malignant disease. The woman had had one hemorrhage since the operation, which was performed a week ago, but it ceased at once on washing the uterus out with hot carbolized water, and the washing brought away a small piece of material such as was removed at the operation. Dr. Dudley had not encountered a similar case, and had had grave apprehensions concerning its possible malignant character.

Dr. Dudley also presented a specimen of

HÆMATOMA OF THE OVARY,

removed one week ago from a patient twenty-two years of age, who had been under his care for one year, during which time he adopted a great variety of general internal treatment, with local internal and external applications; but the woman was not relieved a particle of the constant pain in her left side, which increased in severity with each return of her monthly sickness.

An exploratory incision was made, the left ovary easily lifted and removed, and on opening it there was found a hæmatoma that occupied nearly the entire organ, which was not of frequent occurrence. The patient was convalescing, and, thus far, had not suffered from the pain of which she formerly complained.

Dr. W. M. Carpenter mentioned a case which was almost identical with the one first reported by Dr. Dudley. A woman who had passed the menopause ten or twelve years was profoundly shocked by the death of her husband, and began about two months thereafter to have slight uterine hemorrhage, which continued more or less constantly for more than a year, with one profuse flow, and attended only by backache. The discharge was odorless, and there was neither loss of flesh nor cachexia. Dr. James B. Hunter took charge of the case; found no evidence of disease of the cervix or peri-uterine tissues, nor any evidence of uterine fibroid. He dilated the uterus, and removed a mass of material which, from the description, was like that described by Dr. Dudley. Since the operation there had been no hemorrhage, and the patient, thus far, had remained well.

Dr. Mary Dixon Jones mentioned a case of recurrent uterine hemorrhage, occurring after the menopause, in which she effected a cure by dilating and curetting the uterus.

TUBERCULAR MENINGITIS—AN OLD CASEOUS NODULE IN THE BRAIN.

Dr. L. Emmett Holt presented a specimen with the following history. The specimen was referred to the Committee on Microscopy.

It was taken from the body of a female child, aged four, who died at the New York Infant Asylum after ten days' illness. The parents were healthy, but the mother's family was tubercular. The child was plump and healthy in appearance up to the beginning of the last illness. The onset was gradual, being characterized by listlessness, anorexia, drowsiness, and constipation, but no vomiting or headache.

The symptoms were typical after the first three days. There was abdominal retraction, well-marked *tâche cérébrale*, loss of knee-jerk, slow, irregular pulse, Cheyne-Stokes respiration, opisthotonos, dilatation of the pupils, and semi-stupor, which merged into true coma. For the first four or five days there was almost complete left hemiplegia, with greatly diminished sensibility upon the same side ; this did not disappear, but the opposite side gradually became affected to the same degree.

There were no convulsions, hydrocephalic cry, or vomiting. The temperature was from 100° to 101.8° F., until two days before death, when it ran up rapidly to 104° F. Death took place quietly.

Autopsy, two hours after death. Body well nourished.

Head : The brain was intensely engorged, the convolutions flattened. There were no tubercles or signs of inflammation except at the base, especially abundant over the frontal lobes, in the interpeduncular space, and along the great longitudinal and Sylvian fissures ; here they matted the tissues together quite firmly. Inflammatory products about equally distributed over the two sides. In the left Sylvian fissure, just internal to its bifurcation, was a caseous nodule, the size of an ordinary grape, which seemed to be meningeal in its origin and connections. The ventricles were dilated, each containing about an ounce of clear serum ; ependyma congested, no tubercles seen. Spinal cord not examined.

Thorax: The lungs were healthy; no tubercles. The bronchial glands were cheesy, and as large as beans. The heart-auricles were distended with black clots; no endocarditis; no tubercles in pericardium.

Abdomen: A very few tubercles were scattered over the surface of the liver and spleen; none were found elsewhere, though careful search was made. The intestines were normal, except some recent congestion in the colon. The mesenteric glands were not cheesy.

The other organs were normal, except the kidneys, which showed some cloudy swelling. No disturbance of speech was observed during life.

Dr. W. H. Porter presented a specimen of

STRANGULATED BUBONOCELE

with a peculiar history. The man from whose body it was removed was a waiter, between thirty and forty years of age, in whom developed suddenly the active symptoms of strangulated hernia. When a physician was called he found a large and painful tumor in the right inguinal region, and also a large tumefaction of the scrotum. The man was suffering with intense pain, had vomited stercoraceous matter, and was constipated. The physician applied taxis and caused the rapid disappearance of the scrotal tumor. But the symptoms of strangulated hernia continued, and he made repeated attempts by taxis to reduce the tumor in the inguinal region, but failed. The patient became partially moribund, and in that condition was transferred to the hospital, where he died soon after admission.

At the autopsy the knuckle of intestine presented was found in the inguinal canal. About one pint of bloody serum was found in the peritoneal cavity, and it was also found that the tunica vaginalis had been ruptured. It was believed that through the rupture in the tunica vaginalis the fluid of

THE ACUTE HYDROCELE

had been forced upward into the peritoneal cavity by the taxis. The peritoneum covering the strangulated portion of intestine remained bright and glistening, and there was no evidence of peritonitis.

Dr. Ira Van Gieson presented a specimen of

MULTIPLE DIVERTICULUM OF THE BLADDER, ONE OF WHICH CONTAINED A NUMBER OF SMALL URINARY CALCULI.

The bladder was removed from the body of a patient who died in Charity Hospital. Its inner surface was perforated by five apertures, ranging from 3 to 8 mm. in diameter, leading into irregular-shaped cavities in the wall of the bladder. Back of the urethral orifice, in the posterior wall of the bladder near the median line, was an ellipsoidal diverticulum, 1 by $\frac{3}{4}$ ctm. in diameter, distended by white calculi, over 110 in number, about the size of shot. The calculi caused a slight bulging forward of the mucosa, and the orifice by which the diverticulum communicated with the interior of the bladder was so small that it escaped observation. The right kidney was small, measuring 3 by $1\frac{1}{2}$ ctm. in diameter, and reticulated, but seemed normal. The left kidney contained a calculus in the pelvis, about the size of a pea.

There were no symptoms referable to the bladder. The point of interest was the presence of these little pockets, which favored the formation and retention of small calculi in the mucous membrane of the organ.

The President remarked that the specimen was especially interesting in connection with the one presented by Dr. Northrup. In one case there was hemorrhage by diapedesis, and in the other an accumulation of small vesical calculi in these diverticula in the mucous membrane.

The Society then went into executive session.

Stated Meeting, November 9, 1887.

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

DR. J. S. THACHER, from the Committee on Microscopy, reported that the ovarian tumor presented by Dr. A. P. Dudley, September 28, 1887, showed a stroma of connective tissue quite rich in cells in places, with many alveoli, in some of which could be seen a lining of cylindrical epithelium. The tissue therefore resembled that of a

MULTILOCULAR OVARIAN CYST.

DR. HODENPYL presented, in behalf of a candidate, a specimen of *aneurism of the descending portion of the arch of the aorta.*

DR. J. S. THACHER presented, in behalf of a candidate, specimens of blood which showed the *plasmoideum malariæ.*

DR. LEWIS HALL SAYRE presented, in behalf of a candidate, a specimen of *abdominal tumor*, which was referred to the Committee on Microscopy.

DR. MARY DIXON JONES presented a microscopic section which showed the lesions of

CHRONIC CERVICITIS,

and she raised the question whether the changes affecting the smooth muscle-fibres and the presence of connective tissue might not be causative factors in cervical lacerations.

Dr. Jones also presented four specimens of

TUBES AND OVARIES,

with the history of suffering by the patients and benefit by the operation. In one case there were extensive adhesions; in one, no adhesions; in one the patient ceased menstruating at the age of twenty-five; in three cases the women had not borne children, and in the other two children.

Dr. Jones raised the question of the length of the abdominal incision which was best to be adopted.

DR. WACKERHAGEN presented the amputated limb in a case of

SENILE GANGRENE, INVOLVING THE POSTERIOR REGION OF THE LEFT ANKLE-JOINT,

occurring in the person of Mr. R——, a native of the United States, seventy-eight years of age, and by occupation a farmer. His health had always been good until last April, when he noticed a small, dark-colored spot upon the lower and posterior part of the left ankle. The discoloration gradually spread to both sides of the tendo Achillis, and upward for about three inches above the posterior tuberosity of the os calcis, where the line of demarcation formed, leaving a slough which involved the posterior portion of the calcaneum, the tendo Achillis, and adjoining soft parts. His general health was good. This was his condition when he first saw him, with Dr. J. E. Gregory, on October 13th.

Tonics and nourishing diet, with moderate stimulation, were prescribed. On the day following, the gangrenous slough was cut away with scissors, and the surface thoroughly washed with sublimate solution—1 to 2,000—and dressed with iodoform gauze. These dressings were changed daily. On Tuesday, October 18th, a healthy granulating surface having appeared, the sublimate irrigations were discontinued, and a two per cent. solution of carbolic acid was substituted. On Thursday, October 20th, the granulation appeared of a dusky hue, and as the foot was becoming quite painful it was determined to accede to his wishes, he being anxious to have the affected limb removed.

Amputation was performed on Saturday, October 22d, at the lower third of the thigh, by the circular method. The femoral vein was pouched, and its walls were so fragile that the slight effort in separating it from adjoining structures caused it to tear. Calcareous deposits were found in the walls of the femoral artery at and above the place where it was divided, and it was found necessary to follow it up for a distance of nearly two and one-half inches before a suitable place was reached for the application of the ligature. All vessels were ligated before the removal of Esmarch's bandage; but as there was some oozing from the cut ends of the muscles it was found necessary to apply hot water to the stump, after the method of Dr. Varick. This stopped all bleeding in a few minutes, when the drainage-tubes were placed and the short skin-flaps were united with silk-worm gut. Sublimate irrigation was used very sparingly, boro-salicylic solution being substituted for it during the operation. The edges of the sutured wound were slightly sprinkled with iodoform. Having applied the rubber protective, the stump was dressed with five per cent. iodoform gauze, and covered with sublimate gauze; over this was placed carbolized cotton, and over all a large, fine-wool cushion.

The patient came out of the anæsthesia in good condition, without indication of shock or prostration. He was in a cheerful state of mind, and continued to improve until Thursday, October 27th, when the pulse increased in frequency (90), and the temperature, which had been normal, increased to 100$\frac{2}{5}$° F.; the temperature, however, decreased on the following morning to 99$\frac{1}{2}$° F., but the pulse increased to 100. The tongue became furred, and there was some disposition to diarrhœa. This was readily checked with bismuth.

On Friday, October 28th, the temperature again rose to 100$\frac{3}{5}$° F., and the pulse to 110. On the following morning the dressings were changed for the first time, and we were gratified to note that the wound had almost completely united, and the stump was in every respect in excellent condition. The patient was also generally improved, and so expressed himself. About eight o'clock the same evening the nurse telephoned to Dr. Gregory, who found Mr. R—— rapidly failing. There were involuntary discharges from rectum and bladder. He died at nine o'clock, with symptoms of heart-failure. Post-mortem examination was not permitted.

The specimen showed

CALCIFICATION OF ALL THE ARTERIES, ANTERIOR AND POSTERIOR, TIBIAL, PERONEAL, AND POPLITEAL.

DR. IRA VAN GIESON presented specimens of

MULTIPLE CAVERNOUS ANGIOMATA OF THE LIVER,

removed from the body of a middle-aged woman. In the liver there were about thirty tumors, varying in size from 3 mm. to 1$\frac{1}{2}$ ctm. in diameter. The tumors were of a deep-red color, sharply outlined from the adjacent liver-tissue, and were composed of anastomosing trabeculæ and laminæ inclosing irregular-shaped larger and smaller spaces filled with blood. The surface of the liver was retracted over the superficially situated masses. Some of the tumors were surrounded and intermingled with dense white areas consisting of connective tissue moderately well supplied with small capillaries.

Dr. Van Gieson also presented a specimen of

NEUROMA OF THE SCIATIC NERVE AFTER AMPUTATION OF THE THIGH.

The nodule measured one-half by one-fourth of an inch.

The best illustration and description which he had seen of these tumors was by Krauser and Friedländer, who had described the changes in the peripheral nerves, and in the spinal cord, studied particularly in a case of amputation of the arm, where the nonules were found to be composed almost exclusively of nerve-fibres.

The theory advanced was, that after the nerve was divided in amputation the motor fibres perhaps seek to join the peripheral segment of the nerve which has been removed, and in their wandering, as it were, push against the resisting tissues and form nodules on the proximal end of the nerve.

The President remarked that one point of interest in Dr. Van Gieson's specimen of angioma was the large number of little tumors.

Dr. Ferguson had not seen more than four or five of these growths in a single liver. The largest he had seen was about two and a quarter inches in diameter.

Dr. Carpenter had not found more than three or four; and

Dr. W. H. Porter had not seen more than four or five, and they had been small in every case except one, where the whole liver was involved.

The President referred to a case in which the growth projected from the liver to about the size of the pregnant uterus, and was removed by operation, but with what result was not stated in the report.

Dr. Frank Ferguson presented a

MICROSCOPIC SECTION OF THE STOMACH, SHOWING A RUPTURED ARTERY WITH SUBMUCOSA AND ITS ACCOMPANYING VEIN.

It was from the body of a woman, twenty-two years of age, who died of septic parotiditis, developing in the state of exhaustion due to loss of blood, which showed itself by profuse hæmatemesis and the characteristic evacuations from the bowels. The first hemorrhage occurred three months after an attack of black measles, from which the patient apparently recovered completely.

The case had been reported by Dr. Abbe, in the *New York Medical Journal*, October 29, 1887.

The autopsy was made by Dr. Ferguson, and showed, in addition to marked anæmia of all the organs, a stomach which was normal in size and location; the cardiac and pyloric orifices were normal in size and perfectly free. The mucous membrane was smooth and very pale in color. In the posterior wall, 6 ctm. from the œsophageal orifice, and just to the right of a line drawn parallel to the œsophagus, was an apparent loss of the mucous surface, circular in shape and 6 mm. in diameter. In its centre was a hard, nipple-shaped elevation, and beside this elevation, extending transversely across the implicated area, was a vessel with thin walls partially filled with blood. There was no elevation of the edges of the area, and by the unaided eye it could not be identified as an ulcer. Microscopic examination of the area showed a total loss of the mucous membrane to the extent of the measurements given above. At the base of this ulcer were seen an artery and vein of considerable size.

THE WALLS OF THE VEIN WERE EVERYWHERE INTACT. The hard, nipple-like elevation, already described, was the ruptured wall of an artery. Sections carried along through the entire ulcer showed a rupture of the artery involving one-half of its wall; the other half showed no evidence of disease. A mass of fibrin and blood clung to the ruptured wall, and for a short distance on either side of the rupture the wall was split up and contained fibrin and blood. There was a zone of small round cells surrounding the ulcer, and at the bottom the capillaries were filled with blood and the round cells of inflammation infiltrated the connective tissue. Sections made in other parts of the stomach revealed no lesion of its coats or vessels.

Dr. Ferguson also presented specimens which illustrated

CHRONIC DIFFUSE NEPHRITIS—CARDIAC HYPERTROPHY—LESIONS OF THE CEREBRAL VESSELS—CEREBRAL HEMORRHAGE.

They were removed from the body of a man, fifty-five years of age, a native of Ireland, and by occupation a shoemaker, who was admitted to the New York Hospital, with hernia, on October 31, 1887.

On November 2d he became stupid, breathed stertorous at times, his right pupil was a little larger than the left, and there was impaired sensibility of the right side, but no paralysis. His pulse was slow, strong, and full, of very high tension, and his peripheral arteries were atheromatous and tortuous. He had a well-marked *arcus senilis.* He had neither convulsions nor elevation of temperature. He had paralysis of motion and sensation on the right side. His respiration became Cheyne-Stokes in character. His urine was amber-colored, acid, 1.012, and contained fifty per cent. of albumen to the column, with hyaline, granular, and epithelial casts. His coma gradually deepened, his temperature rose to 103° F., on the afternoon of the 6th. His respiration became more shallow, his pulse more feeble, and he died on the 7th, at 3 A.M.

The autopsy revealed a hemorrhage as large as an ordinary hen's egg, destroying the ganglia at the base of the left cerebral hemisphere. There was no rupture of the floor of the left ventricle. There were numerous smaller extravasations in the neighborhood of the large hemorrhage. The convolutions were distinctly flattened.

THE VESSELS AT THE BASE WERE DILATED AND MODERATELY ATHEROMATOUS.

The smaller vessels and capillaries in the neighborhood of the hemorrhages showed extensive fatty degeneration of their walls, and in many places the perivascular spaces were filled with large granular cells. In places these granular bodies were also seen within the wall of the vessels.

The walls of the smaller arteries were split up, in places, into layers by small round and spindle-shaped cells, rendering the diameter of some of the vessels six to eight times their natural size. While in many places aneurismal dilatation of the smaller arteries was found, there were no distinct miliary aneurisms noticed. These arteries were shown under the microscope, both stained and unstained.

The kidneys were slightly larger than normal. Their capsules were not adherent. Their surfaces were smooth, their cortices were swollen, and their markings were indistinct. The cortex of each organ contained a very great deal of fat.

The renal arteries were dilated and tortuous. Under the microscope their walls were seen greatly thickened. The tubes of the cortex and pyramids were dilated and contained fat. There was much increase in the fibrous tissue.

The heart was greatly hypertrophied. All its valves seemed competent. The muscular tissue was fatty and anæmic. The coronary arteries were dilated and tortuous, but not atheromatous. Under the microscope there was seen increase in the interstitial fat, and the muscle-cells were swollen, granular, and fatty.

FRACTURE OF THE CERVICAL VERTEBRÆ—HEMORRHAGE IN THE SUBSTANCE OF THE SPINAL CORD.

Dr. W. H. Porter presented specimens taken from the body of a man, twenty-eight years of age, who accidentally fell down one flight of stairs and broke off the posterior arches of the third, fourth, fifth, and sixth cervical vertebræ, and at the same time fractured, antero-posteriorly, the bodies of two of these vertebræ.

When the ambulance surgeon reached the spot he found the patient partially paralyzed. He took hold of his head and raised it upward. While doing so he felt the bones crepitate, and the paresis was so much improved that during the next twenty-four hours there was comparatively little loss of sensation and motion. On the second day the paresis returned, gradually increased, and the patient died from paralysis of the diaphragm.

At the autopsy there was found only a slight amount of hemorrhage in the soft parts about the vertebral column, no hemorrhage between the dura mater and the bone, and none

BETWEEN THE PIA MATER AND THE CORD.

But into the substance of the cord there was hemorrhage, in the space covered by two or three vertebræ, which caused the death of the patient.

One interesting point in the case was the fact that there was little or no hemorrhage outside of the substance of the spinal cord. There were apparently multiple hemorrhages entirely within the substance of the cord.

INTRALOBULAR CIRRHOSIS OF THE LIVER—CHRONIC SPLENITIS.

Dr. Porter also presented specimens with the following history: They were taken from the body of a woman, forty years of age, without the history of syphilis or alcoholism. When admitted to the hospital she had marked dyspnœa, and her abdomen was full of fluid. She was tapped at once, and three gallons of fluid were removed. Stimulants and diaphoretics were administered, the general œdema disappeared entirely, and the condition of the patient improved.

Physical examination then revealed double aortic and mitral lesions. The ascites only returned, and the patient died on account of the extensive accumulation of fluid in the abdominal cavity.

The autopsy revealed aortic and mitral insufficiency. There were five gallons of fluid in the abdomen. The spleen was very much enlarged, and its capsule was dense and very much thickened. It weighed twenty-two ounces. The liver was very markedly atrophied. It weighed twenty-nine ounces. Microscopic examination showed intralobular cirrhosis, with pigmentation of the hepatic cells. The central veins were not involved. The kidneys showed the lesions of chronic diffuse nephritis.

SEPTIC PERITONITIS AND ENDOCARDITIS.

Dr. J. Lewis Smith presented specimens from a new-born infant, with the following history:

Lizzie C——, a primipara, was confined on September 21, 1887. Her labor was normal, lasting five hours, and there was nothing unusual in her case before and after her confinement. The baby, a female, was normally developed, weighing eight pounds and one ounce. She had natural evacuations, took the breast readily, and seemed entirely well until the afternoon of September 28th, when she became restless and refused the breast; rectal temperature 101.4° F.; respiration accelerated markedly, and accompanied by an expiratory moan. On September 29th, A.M., temperature 103.4° F.; was very restless; respiration as before; took milk from a spoon; evening temperature 103.6° F. On September 30th, temperature 104° F.; vomiting, restless; respiration 56, and accompanied by an *expiratory moan* when she was moved; pulse counted with difficulty, but not less than 200; slight twitching of extremities on left side. There was no cough, and a careful examination of the chest by percussion and auscultation gave a negative result. It had been maintained by some writers that the *expiratory moan* indicated the presence of some pulmonary disease, as pneumonia or pleurisy, but neither were present in this case. The abdomen was prominent, or distended, soft, not hard, and it gave a dull percussion-sound over every part. Dr. Smith was uncertain, from the feebleness of the child, whether pressure upon it gave pain. The diagnosis of peritonitis, probably of septic origin, was made. On September 30th, evening temperature 104.4° F.; the patient was sinking. Death occurred at 11.15 P.M.

Autopsy, by Dr. E. C. Wendt: Body moderately emaciated; slight rigor mortis. About three ounces of seropurulent fluid, containing abundant flakes of fibrin, escaped from the abdominal cavity. All the abdominal organs were covered by a thick layer of fibrin; liver and spleen completely covered by it, and intestines loosely matted together by it. Left lobe of liver much enlarged, touching the spleen and left kidney; appearance of a section of the liver normal. Umbilical vein pervious; it contained clots of blood and dirty-looking pus, but the umbilicus was of normal appearance and cicatrized. The brain and its meninges seemed normal, unless a little congested. The posterior cusp of the aortic valve was thickened and rigid, and attached to it was a fibrinous mass. The appearance indicated

A LOCALIZED ENDOCARDITIS

at and around the valve; otherwise the thoracic organs seemed normal.

A microscopic examination of the organs was made by Dr. Frank Grauer, of Carnegie Laboratory, who found no evidence of inflammation in the umbilical vein, but thinks, from the history and appearance, that it contained septic matter from which the peritonitis and endocarditis originated of which the infant died. The liver was not only swollen, but under the microscope the peculiar cloudy appearance characteristic of parenchymatous inflammation was observed. The kidneys were normal, the straight tubules containing, as is common in the new-born, beautiful urate-of-sodium crystals. The cardiac muscular fibres were distinctly granular in places. Dr. Grauer's description of the state of the aortic valve was as follows: "There was slight endocarditis, situated at one of the segments of the aortic valve, to which was attached a small thrombus."

SEPTICÆMIA OF THE NEW-BORN

is one of the most severe and fatal maladies of early life. It appears to originate, ordinarily, from decomposing thrombi in the umbilical vein, in which multitudes of micrococci are developed. These organisms, with fragments of thrombi undergoing decomposition, pass along the umbilical vein to its branches which enter the liver, and to the ductus venosus, and finally entering the ascending vena cava, produce a general systemic infection. Disintegrating particles of the fibrinous clots, loaded with micrococci intercepted in the organs, cause embolisms and septic inflammations. Septic inflammations, whether occurring in the interior of organs or upon one of the surfaces as in this case, in which a general peritonitis was developed, are intractable and usually fatal.

Treatment.—It is obviously very important that strict cleanliness be observed in the treatment of the umbilicus of the new-born, since it is supposed that the entrance of microbes through the umbilicus into the umbilical vein causes the putrefactive changes which produce the septicæmia. Care should be taken that the water and sponges employed in bathing the child be free from impurities, that no dirt or foul substance lodge in the umbilicus, and that the abdominal bandage be entirely clean. It might be well to dust the umbilicus with some antiseptic powder, as a mixture of bismuth and iodoform, as a preventive measure.

DR. H. J. BOLDT referred to the report of a similar case in one of the German journals, in which the writer described it as one of septic peritonitis due to suppuration of the umbilical vein.

THE PRESIDENT remarked that in a very considerable number of cases of acute ulcerative endocarditis, micro-organisms were present, particularly the staphylococcus aureus and albus, which readily caused phlebitis as well as peritonitis. He thought there was a possible, perhaps probable, relation between the inflammation of the umbilical vein, in Dr. Smith's case, and the peritonitis and endocarditis, and that the external appearance of the umbilicus did not preclude the existence and progress of phlebitis.

The President presented specimens of

CIRRHOSIS OF THE LIVER, WITH ATROPHY AND CHRONIC INDURATIVE SPLENITIS,

which were interesting in connection with Dr. Porter's specimens. The history of the case was meagre, and for what there was of it he was indebted to Dr. Hubbard, House Physician, First Medical Division in Bellevue Hospital. The patient was an unknown man, thirty-seven years of age, and a native of Germany. On October 18, 1887. he was transferred from the Insane Pavilion to the Hospital. His mental condition prevented the obtaining of any reliable history from him, and he had no friends.

On admission there were moist râles over both lungs. The upper line of hepatic dulness began at the lower border of the third rib in the mammary line. There was marked ascites, and there was some œdema of the feet and legs. After the removal of two hundred ounces of clear serous fluid from the abdominal cavity the area of hepatic dulness seemed to be about normal, while that of the spleen was markedly increased. Seventeen days afterward he was tapped again, and three hundred and thirty-six ounces of cloudy serum were withdrawn. After that his strength failed rapidly, and his temperature, which had been normal, rose, two days before death, to 101½° F., and he died cyanosed on November 8th.

Autopsy.—The peritoneal cavity contained about two litres of turbid reddish serum. The diaphragm on both sides was at the level of the third rib. Heart and lungs normal. Liver was small; its right lobe so much diminished in size as to about equal the left, so that the whole organ was ovoidal in shape. It weighed 1,530 grammes, and measured 25 ctm. in length, 12 ctm. in width at the widest part, and 7 ctm. in thickness. The surface was coarsely nodular, and from its lower surface projected several polypoid and globular masses of liver-tissue which had been constricted and forced out by the new formation of connective tissue. The cut surface showed larger and smaller masses of yellowish liver-parenchyma surrounded by bands of new-formed fibrous connective tissue. The spleen was large, dark, and firm, and its cut surface traversed by slender bands of new-formed connective tissue. The capsule was irregularly thickened. The organ weighed 1,300 grammes, and measured 25 ctm. in length, 12 ctm. in width, and 7 ctm. in thickness. The kidneys and other abdominal viscera were apparently normal.

He presented these specimens, showing not uncommon lesions, because the lesions were both typical and excessive, and because the changes in the liver and spleen had brought these two organs, naturally so dissimilar, to very nearly the same size.

The Society then went into executive session.

Stated Meeting, November 23, 1887.

T. Mitchell Prudden, M.D., President, in the Chair.

Dr. W. P. Northrup, in behalf of candidates, presented a specimen of *laryngeal diphtheria*, and also one of *ulcerative colitis.*

The President presented, in behalf of a candidate, a specimen of *aneurism of the arch of the aorta.*

Dr. Hodenpyl presented a specimen of

CYSTIC ADENOMA OF THE OVARY,

removed post-mortem from the body of a woman, sixty-eight years of age, who died two days after admission to the hospital. The abdomen was greatly distended, and contained a small quantity of bloody serum. The small intestines were adherent to the tumor, and greatly distended with fluid fæces. Attached to the uterus was a small fibroma. The left ovary was cystic. In place of the right ovary was the large tumor presented, which contained several quarts of dark, offensive fluid that contained pus-cells, detritus, and crystals of cholesterine. The liver was also presented, which was considerably deformed, probably from pressure of the tumor and the distended intestine.

Dr. Mary Dixon Jones presented a

UTERINE FIBROMA,

removed by operation from a patient thirty-five years ot age, and the mother of one child about twenty years of age. Her greatest suffering had been during the last six or seven years, and for three days and nights each month she had suffered from severe pelvic pains, with profuse hemorrhage, the flow continuing, in varying quantity, about fifteen days, and leaving her prostrated for the remainder of the month. During the last two or three years she had had profuse menorrhagia.

Dr. Jones saw the patient first in August last. She entered the hospital in October, and under rest, good nourishment, and careful nursing her general condition improved. At the end of three weeks an examination was made, when it was found that the tumor had diminished in size considerably. Bearing in mind that Emmet and others had reported cases in which large uterine fibromata had decreased in size, this plan of treatment was continued. The patient, however, began to get worse—the tumor pressed upon the rectum, etc.—and hysterectomy was performed on November 15th, through an incision five inches in length. The tumor began at the internal os, and was quite low in the pelvis. There were some adhesions upon the left side, and two or three par-ovarian cysts. Before the pedicle could be reached, the tumor was enucleated. A temporary clamp was applied, and afterward Koeberlé's serre-nœud was used. A drainage-tube was employed. The woman made a good recovery and was very comfortable, except that the short pedicle seemed to pull upon the abdominal walls.

Dr. Jones suggested a modification of the operation, by first liberating the vaginal wall, then opening the abdominal wall and removing the entire organ; then closing the abdominal wound entirely and leaving the vaginal opening, as the most natural mode of effecting drainage. She queried whether or not such a tumor could have been benefited by electrolysis. She thought that intra-uterine galvanic cauterization might be more dangerous than the operation.

Dr. H. J. Boldt thought it well to try electricity in these cases, as it would frequently relieve symptoms, although the most successful treatment was doubtless by operation.

Dr. Ira Van Gieson demonstrated microscopic sections of the cortex of the brain and cerebellum which illustrated

GOLGI'S SILVER PROCESS OF STAINING GANGLION-CELLS.

The technique of Golgi's methods (*American Journal of Mental Diseases*, May, 1887) is as follows:

1. *The silver process.*—Small pieces (one to two cubic centimetres) are placed in a two per cent. solution of potassium bichromate (or Müller's fluid), which is increased in concentration at each renewal of the fluid up to five per cent. There is no definite length of time for the hardening process. In summer, portions of the brain are hardened fifteen to fifty days; in winter, five to sixteen weeks. The hardening may be hastened by replacing a part of the Müller's fluid by Erlicki's fluid in increasing proportions, or by conducting the after-hardening in a mixture of eight parts of a two and one-half per cent. solution of bichromate and two parts of a one per cent. solution of osmic acid.

The hardened portions are placed, for twenty-four to thirty hours, in a large volume of a solution of nitrate of silver, which varies in strength according to the duration of the hardening—the more protracted the hardening the stronger the solution—up to one per cent. The silver solution must be changed at intervals until the chromate of silver precipitate no longer forms. The sections are washed in alcohol and cleared first in creasote, and then in turpentine. During the hardening it is advisable to test portions occasionally with the silver solution, for the method is somewhat uncertain in its results.

2. *The sublimate process.*—The same results may be obtained if the hardened pieces are immersed for several weeks in a one-half per cent. solution of bichloride of mercury. This method has the advantage of staining very large masses *in toto.*

These methods stain the ganglion and neuroglia cells, with their intricate network of processes, a deep black color and leave the basement substance nearly colorless.

Remarks.—In interpreting the results of Golgi's methods there is a possible source of error, viz., the tendency of some metallic precipitations not to be strictly confined to the cell protoplasm. The frequency with which certain metallic precipitations occur in spaces and in fluids around the cell protoplasm, in other tissues than the brain, requires great caution in deciding whether some of the delicate black lines connected with the ganglion-cells really indicate the presence of processes.

ACUTE IDIOPATHIC NEPHRITIS IN AN INFANT.

DR. J. LEWIS SMITH presented specimens with the following history. They were removed from the body of an infant, aged thirteen months, an inmate of the New York Foundling Asylum. The baby was wet-nursed until November 1, 1887. In July it had a febrile movement that continued three or four days, without known cause ; at one time the temperature reached 105° F. In August it had pertussis, which ran the usual course. On November 1st the child was apparently perfectly healthy. It was then "farmed out," and was away one week, when it was returned, nearly in collapse, having vomited almost all the nutriment given to it. The stools were infrequent, and were not unhealthy in consistence. There was no evidence of primary cerebral or meningeal disease. The urine was scanty, heavily loaded with albumen, and contained granular casts. Death occurred on November 21st, of probable uræmic poisoning. From November 9th to the 21st the child's temperature fluctuated been 100° F. and 105° F. Dr. W. P. Northrup, curator, made the autopsy, and found that there was moderate pulmonary inflammation, no gastro-enteritis, and no evidence of cerebral disease. The ascending colon near the ileo-cæcal valve was markedly hyperæmic. The kidneys showed the lesions of acute nephritis.

Dr. Smith said that one point of interest in the case was the occurrence of what seemed to be *idiopathic nephritis*, which had been regarded as a rare disease in early life.

The only disease of which there was any history in the case was

PERTUSSIS,

and nephritis could not be regarded as a sequel of that affection. He was inclined to the opinion that idiopathic nephritis in young children was not so frequent as might be supposed from the published cases, and that in a certain proportion of those cases there had been an overlooked mild attack of diphtheria or scarlet fever to which the kidney disease was due. It had been taught that the occurrence of nephritis as a sequel of measles was rare. He had never seen a case in private practice, and thought that if it occurred it must be very mild.

DR. J. C. PETERS had encountered numerous cases of nephritis secondary to scarlet fever, which had been overlooked, and in one patient who was eighty years of age.

DR. W. P. NORTHRUP had met with one case of

ACUTE DIFFUSE NEPHRITIS,

in a child *five months of age*, that had been under such observation as made it certain that it had never had any other disease.

DR. PETERS referred to the high rate of mortality from measles last year and this, according to the records of the Health Department.

DR. SATTERTHWAITE had supposed it to be generally accepted that nephritis was secondary rather than primary, and he had always held that scarlet fever, measles, and diphtheria were the regular precursors of Bright's disease. He had believed that acute parenchymatous nephritis in children was always secondary.

DR. W. H. PORTER said that many patients died with the ordinary clinical symptoms of uræmia, but without either casts or albumen in the urine prior to death. In such cases the kidneys were large, the epithelia were swollen and granular, and unquestionably the organs were unable to eliminate the effete material which they were called upon to remove.

DR. T. E. SATTERTHWAITE presented two microscopic slides from a specimen of

LARYNGEAL CARCINOMA,

with the history of the case and remarks on the *value of the microscope in diagnosis*.

"I think it is an opportune moment to bring before this Society a topic that is now very properly agitating medical minds, viz., the value of microscopic examinations in cases of malignant diseases. And I do so more particularly at the present time, because of a recent discussion at a society meeting in which I took part, and where an effort was made to contrast clinical diagnosis with microscopic, and to the discredit of the latter.

"But now the unfortunate issue of a certain case across the water, belying as it does the printed reports of an eminent pathologist, may with reason set the current back against those who have been working for years past to erect microscopic diagnosis into

A DEPARTMENT OF SCIENTIFIC MEDICINE.

"I therefore take the opportunity of exhibiting two slides, and give the history of a case which furnishes an interesting lesson, from which can be derived valuable corollaries. The history is condensed from the records of the Presbyterian Hospital.

"D. O'L——, aged sixty-three, Ireland, married, entered the hospital February 22, 1886. He had enjoyed remarkably good health until nine months previously, when he swallowed a small fish-bone, which he described as 'thread-like.' Immediately afterward he began to suffer pain in the windpipe, and spit up 'blood and matter.' After two days' suffering the pain abated, so that the bone was suffered to remain undisturbed. Two weeks later pain recurred in the same spot, and after an interval of two months he began to lose his voice. At this time he consulted several specialists, and the solid nitrate of silver in stick form was several times applied to the throat. After about seven months he had some dysphagia, and since that time took no solid food, spoke but little, and gradually lost flesh and strength. About the eighth month he had a severe hemorrhage, losing about a cupful of blood in a few minutes.

"On admission he was found to be suffering a good deal of pain. There was also marked stridor and dyspnœa. Breathing best when lying down. Does not speak above a whisper.

"Laryngoscopic examination showed a swelling of both arytenoids, especially of the left, and of the right ary-epiglottidean fold.

THE TUMOR WAS GRAYISH ON THE SURFACE, WITH SCATTERED SMALL RED SPOTS.

"No paralysis of the cords. Larynx tender on pressure. Further examination showed that the liver was not enlarged. No sternal or tibial tenderness. No nodes. Cervical glands somewhat enlarged. A consultation was held between the hospital surgeons, prominent specialists being also present. Opinions were divided, one-half holding that the affection was inflammatory, due to the lodgment of a foreign body; the other half, comprising the hospital surgeons in attendance, making a diagnosis of

LARYNGEAL EPITHELIOMA.

"On the day following tracheotomy was performed. The laryngeal cavity was made plainly visible, and the new growth was seen to involve, not only both sides of the larynx, but also to extend downward in the wall of the trachea as far as the rings were cut.

"Portions of the growth were then removed for microscopic examination, and the tracheotomy tube was inserted. The operation gave relief, but the patient died three months later, probably of exhaustion from the extension of the disease.

"At post-mortem examination the right lung was found strongly adherent to the chest-walls; the left lung was slightly adherent at base and apex. The heart, lungs, larynx, tongue, and œsophagus were removed *en masse.* The œsophagus was free from disease, except at a point where it was in close relation with the laryngeal disease, where its coats were slightly infiltrated. The inferior half of the laryngeal cavity was occupied by papillary and nodular growths, which had completely or nearly occluded its lumen. The lower part of the growth was in an ulcerated and sloughing condition, and portions of the cricoid and thyroid cartilages had been destroyed by the disease. The walls of the trachea were infiltrated half-way from the larynx to the bifurcation. The cervical glands of the left side were found enlarged. The lungs appeared to be free from secondary deposits. The heart, spleen, kidneys, and liver showed no implications.

"This case is interesting to me at the present time for an especial reason, viz., because the papillary and nodular growths were several times examined in my laboratory, by myself and assistants, and

NOTHING CANCEROUS WAS FOUND;

and yet the post-mortem examination revealed in the enlarged and infiltrated cervical glands the characteristic cell-formations exhibited under the microscope.

"With this case in view, it may be well to emphasize the fact that one cannot be too cautions in the delivery of an opinion in such cases, especially where the papillary or villous growth is the only part that is submitted for examination. And I presume that this opinion is held generally in New York.

"Indeed, I think it is generally admitted that one should never give an opinion in such instances unless the evidences are unmistakably in favor of this or that growth. It often happens that one instantly stumbles upon such microscopic proof that the diagnosis is made at once, and yet one may not be so fortunate, and for very good reasons: (1) At the inception of the disease the pathognomonic evidences are absent, or are so slight that they may be overlooked. (2) There is always a focus of the disease in which these characters are seen after, to the exclusion of more distant parts. (3) In a growth that is growing rapidly and ulcerating, the characteristic tissue may be constantly sloughing off.

"But I would not wish it understood that the statements tend to depreciate the value of the microscope. It has its field, in which it is of paramount value, but it is a limited field; and it should not be relied upon in every instance to the exclusion of clinical signs—and yet it is in this class of cases that the microscope often gives the most brilliant results. But when it fails it is usually because the pathologist is not furnished with a sufficient amount of tissue on which to base his opinion.

"In this connection I would venture to state that, in my humble opinion, the real error in the diagnosis of carcinoma among ordinary competent microscopists is very small. On page 329, vol. iii. of our Transactions, I put it that one per cent. is the figure. A single mistake only occurred in a hundred cases reported by myself, working conjointly with Dr. W. H. Porter. In that instance the specimen sent for examination had been changed by decomposition."

THE PRESIDENT remarked, with reference to the case which all had in mind, that those who had made adverse criticisms on Virchow had overlooked entirely the fact that a benign growth may be followed by a malignant one.

DR. SATTERTHWAITE raised the question,

IS NOT THE MICROSCOPE COMPETENT TO MAKE A DIAGNOSIS WITH REGARD TO THE VARIETIES OF CARCINOMA?

The question was answered in the affirmative by the President and by all those present who had had practical experience in the use of the instrument.

The Society then went into executive session.

Stated Meeting, December 14, 1887.

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

DR. R. W. AMIDON presented, in behalf of a candidate, a specimen of *congenital diaphragmatic hernia.*

DR. HODENPYL presented, for a candidate, a specimen of *epithelioma of the cervix, with suppurative nephritis.*

DR. W. H. PORTER presented, in behalf of a candidate, a specimen of *incapsulated cerebral abscess, secondary to a pistol-wound.*

DR. H. P. LOOMIS presented a specimen of

PRIMARY TUBERCULOSIS OF THE KIDNEY,

with the following history: The patient was a man, thirty-two years of age, who was admitted to Bellevue Hospital, November 17, 1887, after a protracted debauch. His mother died of phthisis. He had had gonorrhœa, but there was no history of syphilis. He had suffered from gastric disorders, due to alcoholism. Six years ago he had pneumonia, and one year later rheumatism. About six months ago, after a protracted spree, he had an attack of what was diagnosticated as "acute Bright's disease," but he says he made a good recovery. About November 17, 1887, he began to have fever, and his temperature was 101° F. His abdomen began to enlarge, there was swelling of the feet, his urine contained albumen, was scanty in quantity, dark-colored, and loaded with urates; there were no casts, but some blood and pus. Bowels constipated, and skin slightly jaundiced. On November 19th he began to have cough, his difficulty of breathing and fever increased, and the physical signs led to the diagnosis of general bronchitis. He grew worse rapidly, and died on December 2, 1887, with increased anasarca and jaundice.

At the autopsy the abdominal cavity contained a large quantity of straw-colored fluid. The heart and pericardium were normal. The left lung was generally adherent; the right lung was firmly bound to the chest-wall in places. On section the lungs were found to be studded with minute, elevated, yellowish nodules, from the size of a pin's-head to a small pea. The bronchial glands were enlarged and pigmented. The left kidney was enlarged, but otherwise normal. The liver weighed seven pounds, and was cirrhotic and fatty.

The cæcum and ascending colon were adherent to the right ureter and kidney, and showed numerous broad tubercular ulcers nearly to the transverse colon. The edges of the ulcers were everted and thickened, and their bases presented a grayish appearance.

The kidney was enlarged, the capsule was non-adherent. The pelvis was dilated; the pyramids were nearly destroyed by the formation of irregularly shaped cavities which also had encroached upon the cortical substance. These cavities had ragged walls and contained yellowish caseous material. Throughout the cortical portion there were scattered small nodules which, microscopically, showed cheesy centres surrounded by an area of lymphoid cells, with numerous giant-cells.

The walls of the ureter were greatly thickened, and infiltrated with tubercles. The walls of the bladder were thickened, and the mucous membrane presented the appearances usually seen in chronic vesical catarrh, but there was no tubercular infiltration.

He regarded the case as one of primary tuberculosis of the kidney; or, better, perhaps, tubercular pyelonephritis.

It was interesting to observe how the tubercular process extended to the intestines, and, lastly, to the lungs, manifesting itself in an eruption of miliary tubercles. Another point of interest was the fact that only one kidney was involved.

Dr. Loomis also presented a specimen of

CONGENITAL CYSTIC DEGENERATION OF THE KIDNEY,

removed by Dr. W. M. Polk from a female child, aged twenty-two months. An enlargement in the right side of the abdomen was first noticed when the child was eight months old, which was painless, and produced no symptoms subsequently except shortness of breath from pressure upward. The child was well nourished. The mass in the abdomen extended from the pelvis to the free border of the ribs upon the right side, and beyond the median line to the left.

Dr. Polk made an incision, three inches long, in the median line, below the umbilicus. It was then found that the tumor was adherent to the vermiform appendix and transverse colon, and that it was behind the peritoneum.

The abdominal incision was enlarged to within one inch of the ensiform cartilage, and the tumor brought out through the opening, when it was found that it had a pedicle about two inches in diameter. The peritoneal covering was incised and peeled off from the growth, and several adhesions were ligatured and cut. The peritoneal covering was stitched to the abdominal wall with catgut sutures, and the incision was closed with silk-worm gut, after introducing a large drainage-tube. The child reacted partially from the chloroform, but died from shock two hours after the operation. The left kidney was apparently normal. There was neither uterus nor ovaries.

The mass weighed five pounds, had irregular shape, and was enclosed in a dense fibrous capsule that sent prolongations inward. The contents consisted of an immense number of cysts, varying in size from those which were microscopical up to the size of an olive.

Microscopically the contents of one of the cysts showed a collection of epithelioid cells that presented the appearance of a uriniferous tubule. Another cyst showed a tuft of capillary vessels which were regarded as part of a Malpighian body.

Dr. Reginald H. Sayre presented a

DISSECTING-ROOM SPECIMEN OF MORBUS COXARIUS.

The disease had not been diagnosticated, and the case had been recorded as one of myelitis. There was no external sinus. There was rupture of the capsule of the joint, with extravasation of pus and perforation of the acetabulum. No pus was found in the rectum. The disease was chiefly acetabular, and the head of the bone was in its normal position. In the seventy-four cases in which his father had performed exsection, the head of the bone had not been out of its place, and he had therefore concluded that the so-called *dislocation* of the third stage of hip-joint disease was due to rough manipulation.

Dr. Hodenpyl presented a heart that showed

ACUTE PERICARDITIS, WITH THE PRODUCTION OF FIBRIN AND PUS.

There was no history of the case. At the bottom of the pericardial sac was a thick layer of cheesy material. The inner surface of the pericardium and the outer surface of the heart were covered with a thin layer of fibrin.

Dr. Hodenpyl presented a second heart, that showed

CHRONIC ENDOCARDITIS.

The organ was about normal in size. The tricuspid valve was greatly thickened, and it was on that account especially that the specimen was presented. The only history was that the woman was brought into the hospital with extreme dyspnœa and cyanosis, and died soon afterward.

Dr. Hodenpyl presented a third heart, which showed

MARKED HYPERTROPHY AND DILATATION, WITHOUT FATTY DEGENERATION OF THE MUSCULAR FIBRES.

The cavity of the right ventricle was very much enlarged, and the cavity of the left ventricle to a less extent. The kidneys showed the lesions of chronic diffuse nephritis. The heart was about twice its normal size.

Dr. Van Santvoord said that the absence of degeneration of the cardiac muscle was worthy of notice. He thought it was generally believed that when there was heart failure from any cause, degeneration of the heart muscle was commonly present, but he believed that the contrary was quite often the case, and that when from the clinical history of the case there was manifest heart failure, post-mortem frequently showed that there was no lesion of the muscle itself.

Dr. W. H. Porter directed attention to

PERFORATION OF ALL THE CUSPS OF THE AORTIC VALVE

in the specimen presented, and said that according to his experience similar openings were present in forty per cent., at least, of aortic and pulmonary segments. He had seen these perforations as early as the second or third year of age, and believed them to be congenital defects. He thought the specimen was one of those hearts which illustrated that the physical signs of aortic regurgitation might occur without the rational symptoms of that lesion.

Dr. Van Santvoord remarked that the openings referred to he had frequently seen, and, as in the specimen presented, they had been immediately under the very edge of the leaflet. It certainly seemed to him that they would not prevent complete closure of the valve, as they were above the line of contact when the cusps were brought together.

The President asked Dr. Porter if in the percentage of cases he had mentioned there was corresponding cardiac hypertrophy.

Dr. Porter replied that the amount of hypertrophy depended upon the size and number of the openings. If they were large there would be some hypertrophy, but in many cases he had seen the diagnosis of aortic regurgitation made and had found only this physical condition at post-mortem to explain it.

Dr. Van Santvoord thought that if this condition existed in as high as forty per cent. of hearts, evidence of regurgitation at the aortic orifice should be more common than it was.

The President said that he had examined the heart with Dr. Hodenpyl, and it had seemed to him that the perforations were so small and so near the edge of the leaflets that they would be covered when the valve was closed, and that the kidney lesion was the more plausible explanation of the hypertrophy.

DR. VAN GIESON presented an

ABSCESS OF THE LIVER

which was interesting chiefly because during life there was tympanitic resonance over the sac which made it appear that the liver was reduced in size.

Dr. Van Gieson also presented a specimen of

ANGIO-SARCOMA OF THE LUNG, WITH CALCIFIC DEPOSITS,

removed from the body of a middle-aged woman who had an amputation at the knee five years ago for a tumor which was not diagnosticated.

One of the lungs contained a few whitish nodules, some partially calcified, varying in size from a few millimetres to two or three centimetres in diameter. At the apex of the lung there was a nodule larger than the others (five centimetres in diameter), and more markedly calcific.

Microscopically the nodules consisted of areas which presented the characteristic appearance of angio-sarcoma, and of other areas made up of connective-tissue trabeculæ, partially calcified, enclosing oval and polygonal cells.

DR. L. EMMETT HOLT presented a tumor of the umbilicus which he regarded as

A PERSISTENT MECKEL'S DIVERTICULUM, COVERED WITH MUCOUS MEMBRANE.

DR. J. S. THACHER presented an

ANEURISM OF THE THORACIC AORTA.

The patient did not have any special symptoms, except some epigastric pain, and was treated for colic. There were found nearly two quarts of coagulated blood in the connective tissue outside of the aneurismal sac. The heart showed a very large papillary muscle, which illustrated an occasional cause of cardiac murmur.

DR. LEWIS HALL SAYRE presented a specimen for which he was indebted to Dr. McKahn. It consisted of

THE HEAD AND NECK OF THE FEMUR,

removed by operation from a boy, five years of age, with good family history, who, three years ago, was taken with disease of the right hip-joint, from which he recovered with anchylosis in about eighteen months. Afterward an attempt to break up the adhesions was followed by inflammation, and in about six weeks an abscess made its appearance on the outer aspect of the thigh.

At that time the limb presented the deformity usually seen in the third stage of hip-disease. The abscess was opened and drained, and it was then found that there was an abscess on the inner aspect of the thigh, and when that was opened the finger could be passed through the capsule, and the characteristic gelatiniform material readily removed.

Exsection was performed last Thursday by Dr. McKahn. The periosteum was very much thickened, and had considerable new deposit of bone in it. The operation was performed antiseptically, and the patient was doing well. The gelatiniform substance had been submitted to Dr. Biggs for examination, with reference to tubercle-bacilli.

THE PRESIDENT presented a specimen of

RUPTURE OF THE HEART THROUGH THE RIGHT VENTRICLE, AS THE RESULT OF GENERAL FATTY DEGENERATION FROM ATHEROMA OF THE CORONARY ARTERIES.

For the epitomized clinical history he was indebted to Dr. W. N. Hubbard, House Physician, First Medical Service, Bellevue Hospital.

A woman, forty-five years of age, had been under treatment for Bright's disease some months ago. She had then complained of "trouble with her heart," and had not been well since. A couple of days before admission to the hospital she became worse; had shortness of breath and a sense of oppression about the heart.

She was admitted on December 7th, in the forenoon, cyanosed, with cold extremities, delirious, restless, and very thirsty. Respiration, 34; pulse, 150, and very irregular and feeble; temperature, 100.6° F. She was unable to sit up, but when propped up by a head-rest the dyspnœa and cyanosis were somewhat relieved. She complained of great pain about the heart. The chest-resonance was good; there were many coarse râles. The area of cardiac dulness was increased; no murmurs could be heard.

She slept a little at intervals during the following night, but was most of the time very noisy and restless. She seemed to develop œdema of the lungs, and died about twenty-four hours after admission.

Autopsy.—The body was well nourished, pale, lips blue. General appearance of abdominal cavity and viscera, normal. Right pleural cavity empty; left contained about five hundred centimetres of bloody serum.

Heart.—Area of exposed pericardium very large. Pericardial sac contained about five hundred centimetres of fluid blood. When this was removed a small triangular opening was seen near the apex of the right ventricle, through which oozed dark fluid blood. This opening communicated directly with the cavity of the right ventricle. The cavities of the right heart contained fluid blood, and voluminous, soft, ragged red clots, entangled among the papillary muscles, and adherent to the walls; valves normal. The left ventricle was firmly contracted, and the left cavities were moderately dilated and nearly empty. The mitral valve was moderately thickened and contracted at the edge, and the attached chordæ tendinæ were shortened. The walls of the coronary arteries were everywhere moderately atheromatous and fatty; but not especially so in the branch supplying the region of rupture. The entire heart muscle was pale and flabby. A small triangular tongue of heart muscle lay valve-like across the opening at the seat of rupture. Microscopically the heart muscle, in all parts of the organ, was the seat of marked, though not excessive, fatty degeneration and chronic interstitial myocarditis; but the fatty degeneration was not more marked at the seat of rupture than in other parts of the heart. The aorta was, throughout, the seat of extensive atheroma and fatty degeneration, and showed patches of calcification.

Lungs.—Both showed emphysema, chronic bronchitis, and moderate œdema, while in both lower lobes, posteriorly, were several hemorrhagic infarctions, from the size of a filbert to that of a small hen's egg. In the branches of the pulmonary artery leading to the two of these infarctions which were dissected, dense, occluding fibrinous clots were found.

All of the abdominal viscera were congested; while, developed to a moderate degree, were found the lesions of chronic interstitial hepatitis and chronic diffuse nephritis, the kidneys being slightly atrophied.

The case appeared to him of interest because the patient apparently lived for several hours after the occurrence of the rupture; because the rupture was at the apex of the right, and not of the left ventricle, as is more usually the case; and finally, because the rupture was that of a universally fatty heart, and not due to a circumscribed muscular lesion, dependent upon a local obstruction of a branch of the coronary artery, as it had been in most, if not all, of the ruptured hearts recently presented before this Society.

Dr. John C. Peters had presented a similar specimen several years ago, from a man forty-five years of age, and in whom there had not been any sign of cardiac disease until an attack of angina occurred. In that case there was almost complete occlusion of one coronary artery.

The Society then went into executive session.

Stated Meeting, December 28, 1887.

WILLIAM P. NORTHRUP, M.D., VICE-PRESIDENT, IN THE CHAIR.

DR. JOHN J. GRIFFITHS presented two specimens, which illustrated

CEREBRO-SPINAL MENINGITIS WITH PUS AND FIBRIN IN THE CEREBRAL VENTRICLES.

CASE I.—Theresa C——, aged five months, was born in the New York Foundling Asylum, and had been out to a wet nurse. On November 8, 1887, she was brought to the Out-door Department, with a history of constipation and repeated convulsions. She was pale, restless, and had a moderate cough.

She was returned to the house with a diagnosis of meningitis.

Dr. Hopkins, the House Physician, furnishes a history of subsequent repeated convulsions, obstinate constipation, trismus, and unilateral convulsions. No opisthotonos. The symptoms continued four days, the patient dying on November 12th.

Autopsy by Dr. W. P. Northrup: The body was poorly nourished; hair worn off the occiput from constant rolling of the head; no eruption.

Brain.—A thick layer of greenish pus and fibrin was spread over the convexity and base, enveloping it everywhere, and extending down and enveloping the cord. The lateral ventricles were dilated to twice their normal size, and contained an abundant exudate of pus and fibrin, which also extended into the third and fourth ventricles. The central canal of the cord was not dilated. The floors and walls of the ventricles were slightly reddened, vascular, and softened.

CASE II.—Josephine S——, aged four months and five days, was brought to the Out-door Department of the same institution on December 1, 1887, with a history of convulsions, opisthotonos, constipation, and left convergent strabismus. She manifested the same symptoms for the following sixteen days, and died, December 17th.

Autopsy by Dr. W. P. Northrup forty-eight hours after death: The body was extremely emaciated; no eruption.

Brain.—On opening the skull the convolutions were seen to be distinctly flattened and dry. There was no exudate upon the vertex, lateral surfaces, or base. The ventricles were dilated to three or four times their normal size, and within them were thick masses of pus and fibrin exudate, extending from the lateral into the third and fourth ventricles. The cortex was not softened; neither was the corpus callosum.

There was moderate softening of the walls of the ventricles, especially the floors.

At the level of the floor of the fourth ventricle, posteriorly, the inflammatory process seemed to have communicated with an area of cerebellar meningitis, which, in turn, extended down the cord as an ordinary spinal meningitis.

The area of cerebellar meningitis was about the size of a silver half-dollar. The central canal of the cord was not dilated.

The pus and fibrin exudate was thus found within the ventricles, upon a limited area of the cerebellum, and investing the medulla and cord.

In reviewing these cases it will be seen that both children were between four and five months old; both were wet-nursed; both females; and both gave symptoms of constipation and convulsions.

The point of special interest is thought to reside in the extensive pus and fibrin exudate in the ventricles.

In Case I. the exudate in the ventricles was associated with extensive exudate over the entire convexity of the brain and about the cord.

In Case II. the exudate in the ventricles was associated with a spinal meningitis, the two forming a continuity of lesions through the thin wall at the floor of the fourth ventricle at its lowest extremity (foramen of Magendie).

In neither case was there any dilatation of the central canal of the cord.

Dr. Northrup remarked that these two brains were the first cases in which he had seen the ventricles involved. He had seen them contain serum in moderate amount, but without dilatation. In tubercular meningitis the ependyma is frequently involved, small, discrete tubercles being easily demonstrated upon it, the ventricles being at the same time dilated and containing serum. This condition could not be called rare.

In cerebro-spinal meningitis, according to the literature of the subject, the ventricles may be dilated and contain sero-pus. This he regarded as rare.

In a moderate search through the literature he had not met with the description of any case in which the lesion had avoided altogether the convexity of the cerebrum and cerebellum, and expended itself in the ventricles of the brain and upon the meninges of the cord.

The sero-pus of the ventricles seemed to have its origin in the vessels of the velum interpositum, which are continuous with those of the pia mater.

DR. A. BROTHERS presented a specimen of

CLEFT TONGUE,

with the following history: Amelia H——, aged one month, was referred to him by Dr. C. E. Denhard. At birth the midwife noticed a cleft in the palate, which has interfered with the proper feeding of the infant. Nursing was impossible, and milk poured into the child's mouth was returned through the nostrils. Although well-developed at birth it has steadily pined away, and when he saw it, on December 27th, it was evidently dying from marasmus. During the past few weeks it had suffered from diarrhœa, for which no physician was consulted. There were two older children in the family, but they were normally developed.

The specimens showed a fissure in the soft palate extending up to the hard palate, and a cleft one-eighth of an inch deep at the tip of the tongue. The lungs were œdematous, and showed hypostatic congestion at the bases. The heart was normal. The liver was very much congested. The kidneys (only one was removed) showed a small cavity, evidently a cyst.

There was a marked irregularity in the alveolar portion of the inferior maxilla, as though the gums were deficient.

The case was interesting on account of the lack of complete closure of the apex of the tongue, which he thought was quite rare.

Fissure of the palate was of interest because the child was likely to die from lack of nutrition. Had this child been properly fed from the beginning, attention having been paid to getting the milk well down into the œsophagus, so that it could not return through the nose, its life might probably have been saved.

DR. LEWIS HALL SAYRE presented

THE HEAD OF A FEMUR REMOVED BY EXSECTION

at St. Vincent's Hospital. H. M. McK——, thirty years of age, a resident of Illinois, remarkably active and athletic, while being initiated in a secret society in May, 1883, was pressed upon by several men while he was bent over, causing very severe pain in the lumbar and dorsal regions, and making him fall to the floor in great agony. He recovered in a short time, and went through with the ceremony. On the next morning he felt a sensation of itching along the left side of the back, and two weeks later had pain in the right groin, and could not walk without the aid of a cane or crutch, because he was unable to bear any weight upon the right leg.

The case was diagnosticated as rheumatism, and the patient resided in Colorado and Mexico two years and four months, where he underwent treatment, but without benefit.

During this time he was always compelled to raise himself on his hands whenever, from any cause, the street-cars jolted. He then began to notice that he was getting out of shape, and went to a gymnasium for the purpose of correcting that tendency. While swinging suspended by his hands he always felt better, but whenever he came down upon his heels he suffered great pain, and, therefore, he always lowered himself very carefully. In the meantime his physicians insisted that his trouble was rheumatism.

Up to this time no knuckle had been detected in his back, although he was aware that he was

TWISTING SIDEWAYS TO THE RIGHT.

While riding on horseback he had to raise himself from the saddle by his hands whenever the horse jumped over any obstacle. In 1886 he had a brace applied, but was unable to wear it. A plaster-of-Paris jacket was also unsuccessfully applied, although it gave him some comfort. He then had another brace applied, but was unable to wear it, and finally came to Dr. Sayre for a plaster jacket, because the first one put on in Chicago by Dr. Gunn had afforded him some relief. That was on April 21, 1887, and he was then walking with a pair of crutches, and his right leg was flexed and abducted. Careful examination revealed morbus coxarius of the right side, in addition to the spondylitis of the lumbar vertebræ, which had become quite prominent. Partial suspension gave him instant and complete relief from pain. Two days afterward a "plaster-of-Paris jacket" was applied which, he said, gave him more comfort than anything he had worn. But when his back had been made easy, he felt the pain in his hip, which he had not thought of before. A weight and pulley were then applied to the limb, and the pain in his hip was relieved. A short hip-splint was then adjusted during the day, and he felt so comfortable that he left for his home in Texas, and was able to attend to his business, which required him to travel many thousands of miles.

He returned a few weeks ago, with his back nearly well, but his hip was worse. He had worn the short splint only one month. He had occasionally used extension at night; but, without the splint during the day, the disease had progressed until exsection was indicated, and the operation was performed, December 27th.

One month ago the patient's urine contained large quantities of albumen and pus, but they disappeared. The right limb was adducted, flexed at an acute angle with the thigh, and everted. There was intense pain on pressure of the head of the femur into the acetabulum. The head of the bone contained an abscess. The acetabulum was extensively carious.

Before the operation a peculiar clicking noise could be produced in the joint by manipulation, and it was found that the acetabulum contained a loose piece of cartilage that articulated with a corresponding eburnated surface upon the head of the bone, and gave rise to the peculiar sound.

Dr. Brothers asked if it was common for spondylitis and hip-joint disease to occur at the same time in one patient.

Dr. Sayre said that it was not infrequent. He had seen several cases.

The Society then went into executive session.

Stated Meeting, January 11, 1888.

T. MITCHELL PRUDDEN, M.D., PRESIDENT, IN THE CHAIR.

THE PRESIDENT referred to the great and irreparable loss which the Society had met with in the death of its Secretary, Dr. Wesley M. Carpenter. Dr. Carpenter had been ill for some time, but with his characteristic fortitude he had kept his ailments largely to himself. Probably the last work that he had done had been for this Society. He attended a meeting of the Board of Trustees, at the house of Dr. John C. Peters, on the evening before his death, and although apparently not feeling at all well on his return home, he had carefully written out the minutes of the Board meeting before retiring. This was not the time to express the Society's appreciation of Dr. Carpenter's work or its admiration of his character. The duties and tasks which his hands had dropped must now be taken up and carried forward as best they could.

DR. G. C. FREEBORN was then elected Secretary *pro tempore.*

DR. J. S. THACHER, from the Committee on Microscopy, reported that the abdominal tumor presented by Dr. Lewis H. Sayre, on behalf of a candidate, December 9, 1887, showed a structure consisting of fibrous connective tissue, which was, in a few small spots, rich in small round cells. It should therefore probably be classed as a fibroma.

DR. IRA VAN GIESON presented a specimen of

MULTIPLE TUMORS FROM A DOG.

There was a brownish-white, dense nodule, about half an inch in diameter, just above the left external canthus, composed of loose fibrillar connective tissue, with numerous pigmented connective-tissue cells. Some of the mesenteric glands were enlarged, and contained soft white nodules consisting of closed, thin-walled blood-vessels surrounded by clusters of cuboidal and polygonal cells, which in places were swollen and filled with vacuoles. The anatomical diagnosis was: Pigmented fibroma of the brow; angio-sarcoma of the mesenteric glands.

DR. HODENPYL presented a specimen of

VERRUCOUS ENDOCARDITIS WITH ASSOCIATED LESIONS,

removed from the body of a woman forty years of age. The patient had extensive general anasarca. The pericardial sac contained four ounces of clear serum. The heart weighed five hundred grammes. There was dilatation with hypertrophy of the left ventricle. The aortic valve was much thickened and in places eroded, and some vegetations were seen on the free surface of the valve. The mitral valve was thickened and stenosed, and very rigid. There was a slight degree of calcareous degeneration in the aorta.

The left lung was enlarged and consolidated in its upper two-thirds, and on section presented a brownish-red color. The liver, which was not shown, was cirrhotic. The kidneys were enlarged and hard. The spleen was enlarged and hardened, and in one portion showed a white infarction. No bacterial examination of the heart-vegetations had been made.

THE PRESIDENT said that not many cases of this kind had been shown to the Society of late, the specimens having been mostly of the ulcerative variety.

The President referred to a report of fourteen cases of endocarditis, seen in Vienna, in which an

EXAMINATION FOR BACTERIA

had been made. Micro-organisms had been found in twelve of these cases. These were all instances of ulcerative endocarditis, but the two cases in which no bacteria were found were examples of simple vegetative endocarditis. It would seem, from the results of medical research at the present time, that bacteria are not apt to be present in cases of simple verrucous endocarditis. The bacteria most commonly found were the streptococcus pyogenes and the staphylococcus pyogenes aureus. In some cases, in which pneumonia was present, Weichselbaum had found the pneumococcus. One or two other varieties of bacteria had also been found. He further suggested the advisability of injecting the valves in these cases. The heart-valves were not vascular, except at their bases. In vegetative endocarditis, however, the valves had been found to be vascular, but the vessels were very minute and their presence could be demonstrated only by careful injection.

DR. NORTHRUP recalled the fact that the President had examined a case of ulcerative endocarditis in a child last summer, and had found bacteria present.

THE PRESIDENT said that the micro-organisms found in this case were slender bacilli. The specimen had been kept in Müller's fluid for some time during the warm weather, and he feared that possibly this fact might account for the presence of the bacilli, which may have worked their way into the large thrombus on the valves. There had, however, been two or three apparently similar cases reported in which this possible source of error had not been present.

Dr. E. C. Wendt had never heard of a case in which bacilli had been found in this form of endocarditis. He thought it was well always to bear in mind the necessity for quick work in these examinations, as mistakes might readily occur through the intrusion of bacteria from the air.

Dr J. West Roosevelt presented a specimen of

PYO-PNEUMOTHORAX DEPENDENT UPON PHTHISIS.

The specimen was removed from the body of a young man who died in Bellevue Hospital. He had suffered from the ordinary symptoms of phthisis for eight months before his death. He gave no history of sudden pain in the chest, or of shock such as usually follows perforation of the lung. The points of interest were the rather peculiar physical signs and the curious lesion. Two days before his death the patient had tympanitic resonance over the lower part of the right chest, with amphoric breathing and voice, metallic tinkle, and a marked succussion sound. To the bottom of the chest, behind, bubbling and subcrepitant râles were plainly audible. The autopsy showed, in the left lung, numerous discrete yellow nodules, the size of a pea.

Between the base of the left lung, the diaphragm, and the costal pleura was a cavity lined with a shreddy, whitish pellicle, and containing about one and a half pint of bad-smelling pus. The abscess-cavity extended upward behind the lung as a narrow channel, alongside the vertebræ, to within three inches of the apex of the lung. This channel passed between the layers of adherent pleura. At the upper part there was a space about two inches square, which was composed of pulmonary tissue, which was not covered with the pellicle. The lung-tissue was eroded and had a pitted appearance, and at this point there was communication with one of the bronchi. The eroded pulmonary tissue looked very like an old tubercular cavity.

The speaker called attention to the fact that, although the cavity contained considerable pus, it gave tympanitic resonance to the very bottom, and also beneath the fluid-level subcrepitant râles were audible. He had heard the

SUBCREPITANT RÂLES BENEATH THE FLUID-LEVEL

in several cases of pleurisy with effusion, and in one case, where the chest was full to the top, these râles were audible to the very bottom. How such sounds were produced was not clear to him. The tympanitic resonance heard seemed to have been caused by the cavity, and it was probable that, even when percussion was made over the fluid, the tympanitic sound from the air above it masked the dulness.

Another point of interest was the question whether the pyo-pneumothorax had been produced by the rupture of a cavity within the lung, or whether an empyema had first occurred, and had then burrowed up behind the lung and perforated it. This point he was unable to decide.

DR. NORTHRUP asked what explanation the speaker had to offer for absence of dulness at the base of the lung.

DR. ROOSEVELT replied that he saw no other explanation than that the fluid in the pleural cavity and the solidified lung offered a good conducting medium through which the resonance from some dilated bronchi was transmitted. A somewhat similar effect was noticed when a portion of the intestine was known to be filled with fluid, yet a tympanic resonance was transmitted through it.

DR. J. C. PETERS called attention to Skoda's work, in which an attempt was made to explain these acoustic puzzles.

The Society then went into executive session.

The following officers were elected for the year 1888:

President: William P. Northrup.
Vice-President: Frank Fergusson.
Secretary: Walter Mendelson.
Treasurer: J. H. Hinton.
Editor: J. C. Peters.
Committee on Admission and Ethics: J. C. Peters, W. H. Porter, J. Emmet Holt, J. West Roosevelt, George R. Lockwood, Jr.
Members of the Publication Committee: George C. Freeborn, W. R. James.

INDEX.

ABSCESS of the brain, 133
        incapsulated, 190
    of the lung, 97, 147
    of the liver, 194
    peri-urethral, 146
    without suppurative bacteria, 80
Adenoma, multiple, of the kidney, 109
    cystic, of the ovary, 182
Aneurism, of the arch of the aorta, 173, 183
    femoral, 129
    miliary, of the brain, 77
    popliteal, 127
    of the thoracic aorta, 155
Angiomata, multiple cavernous, of the liver, 175
Angio-sarcoma of the lung, 194
    of the mesenteric glands, in a dog, 202
Anthracotic pneumonia, 86, 90
Anthracosis pulmonum, 118
Aortic stenosis, 91
Atelectasis, 124

BACTERIA in endocarditis, 205
Bicuspid valves, insufficiency of, 91
Bile-duct, carcinoma of, 147
    tuberculosis of, with dilatation, 125
Bladder, carcinoma of, 101
    diverticulum of, 167, 168
        containing calculi, 171
    ulcers of, 147, 167
Bony excrescences of the frontal bone, 139
Brain, abscess of, 133
    miliary aneurisms of, 77
    miliary tubercles of, 105
    gunshot wound of, 91
    tuberculosis of, 146
Bronchial gland, developing tubercle in, 121
Bronchial diphtheria, 121
Broncho-pneumonia, 136
    persistent, 85
Bubonocele, strangulated, 171

CÆCUM, invagination of, 141
Calculus, renal, 119
Calculi in diverticulum of the bladder, 171
Carcinoma of the bladder, 101
    of the bile-duct, 147

Carcinoma of the head of the pancreas, 147
  of the larynx, 186
  of the liver, 147
  of the stomach, 157
  of the uterus, 88, 90, 116
Cerebro-spinal meningitis with pus and fibrin in the ventricles, 197
Cerebral hemorrhage, 125, 127
Cervical vertebræ, fracture of, 178
Cervix uteri, epithelioma of, 190
Chronic cervicitis, 112
Chronic ovaritis, 112
Cirrhosis of the liver, 91, 108, 179, 181
Clark, Alonzo, memorial note on the death of, 161
Cleft tongue, 199
Committee on Microscopy, reports of, 90, 101, 108, 146, 167, 173, 186
Congenital absence of right kidney, 147
  cystic degeneration of the kidney, 191
  umbilical hernia, 154
  diaphragmatic hernia, 190
Conjunctiva, primary epithelioma of, 144
Cranial cavity, foreign body in, 102
Cyst, dermoid, by inclusion, 133
    of the ovary, 155
  echinococcus, of the liver and peritoneum, 160
  follicular, of the ovary, in a dog, 80
  multiple, of the ureters, 87
  of the ovary, 137

DERMOID cyst, by inclusion, 133
  of the ovary, 155
Diaphragmatic hernia, congenital, 190
Diphtheria, laryngeal, tracheal, and bronchial, 121
  laryngeal, 183
Diverticula of the bladder, 167, 168
Diverticulum of the bladder, containing calculi, 171
  Meckel's, 194

ECHINOCOCCUS, hooklets of, 146
  cyst of liver and peritoneum, 160
Elastic fibres, methods of staining, 82
Empyæma, circumscribed, 132
Endocarditis, chronic, 192
  septic, 179
  bacteria in, 205
  verrucous, 202
Epithelioma, primary, of the conjunctiva, 144
  of the cervix uteri, 190

FATTY degeneration of the heart, 123, 127
Femoral artery, ligation of, under cocaine, 127
Femur, excision of the head of, 94
  intracapsular fracture of the neck of, 155
Fibroid of the uterus, 104, 183
  sloughing, of the uterus, 88
Fibroma of the abdominal cavity, 173
  intracanicular, of the mammæ in a dog, 80
  pigmented, in a dog, 202
  of the uterus in a dog, 80

Flocculus of the cerebellum, tubercular nodule in, 106
Foreign body in the cranial cavity, 102
  in the eye, 87
Frontal bone, excrescences of, 159

GALL-STONES, multiple and retained, 93
Gangrene of the lung, 147
  senile, of the ankle, 173
Golgi's silver process of staining ganglion-cells, 184

HÆMATOMA of the ovary, 169
Hæmaturia, 147, 148
Heart, double mitral orifice of, 77
  enlargement of right, 83
  fatty degeneration of, 123, 127
  hypertrophy of, 177, 193
  rupture of, through the right ventricle, 195
Heart-valves, insufficiency of, 91
Hemorrhage, cerebral, 177
  into the floor of the fourth ventricle, 125
Hernia, congenital diaphragmatic, 190
  congenital umbilical, 154
Herxheimer's method of staining elastic fibres, 82
Hooklets of echinococcus, 146
Hypertrophy of the heart, 83, 177, 193

ILIUM, ostitis of, 95
Intracapsular fracture of the neck of the femur, 155
Intubation of the larynx, 121
Intubation-tube, calcareous deposits on, 151, 167
Intussusception, 141

JAW, lower, removal of, 140
Jennings' solution for transfusion, 129

KIDNEY, atrophy of, with dilated pelvis, 105
  calculus of, 119
  congenital absence of right, 147
  congenital cystic degeneration of, 191
  diseased, of the horse, 136
  misplaced, 104
  multiple adenoma of, 109
  primary tuberculosis of, 190

LAENNEC, globular thrombosis of, 139
Laryngeal carcinoma, 186
  diphtheria, 121, 183
  epithelioma, 186
Leptomeningitis, acute, 77
Ligation of the external iliac artery under cocaine, 129
  of the femoral artery under cocaine, 127
Little's solution for transfusion, 129
Liver, abscess of, 194
  carcinoma of, 147
  cirrhosis of, 91, 108, 179, 181
  echinococcus cyst of, 160
  gunshot wound of, 115
  healed gunshot wound of, 114

Liver, multiple cavernous angiomata of, 175
  parenchymatous metamorphosis of, 93
  rupture of, without hemorrhage, 115
Lung, abscess of, 97, 147
  angio-sarcoma of, 194
  gangrene of, 147
  hemorrhagic infarction of, 83
  stab wound of, 132
Lustgarten's method of staining elastic fibres, 82

MEASLES followed by pneumonia, 124
Meckel's diverticulum, 194
Meningitis, tubercular, 170
Metritis, 85
Middleton-Goldsmith lectures, 1
Miliary tubercles of the brain, 105
  aneurism of brain, 77
Mitral orifice, double, 83
Morbus-coxarius, 194, 199
Myoma, pendulous, of the uterus, 161
  of the stomach, in a dog, 80

NEPHRITIS, acute idiopathic, in an infant, 185
  chronic diffuse, 108, 177
  parenchymatous, 136
  suppurative, 190
Neuroma of the sciatic nerve after amputation, 175
New staining fluid, 107

ŒSOPHAGUS, malignant stricture of, 80
  stricture of, 99
Oöphoritis, chronic interstitial in a dog, 80
Ostitis of the ilium, 95
Ovarian tumor, large sac of, 153
Ovaritis, chronic, 112
Ovary, cystic degeneration of, 183
  dermoid cyst of, 155
  follicular cyst of, in a dog, 80
  hæmatoma of, 169
  malignant disease of, 151
  multilocular cyst of, 173
  prolapsed cystic, 101

PACHYMENINGITIS, chronic, 77
Pancreas, carcinoma of the head of, 147
Panophthalmitis, from injury, 87
Parotid region, tumor from, 103
Pericarditis, 143
  acute, with production of fibrin and pus, 192
  septic, 179
Perimetritis, 85
Peritonitis, chronic, 142
  traumatic, 143
  tubercular, 108
Peritoneum, echinococcus cyst of, 88
Peri-urethral abscess, 146
Pharynx, tuberculosis of, 88
Phosphorus-poisoning, 199
Pigmented fibroma in a dog, 202

Plasmoidium malariæ, 173
Pleuro-pneumonia in an infant, 110
Pneumonia, abscess, gangrene, 147
    acute anthracotic, 86, 90
    broncho-, 136
        persistent, 85
    following measles, 124
    interstitial, 91
    parenchymatous, 124
    pleuro-, in an infant, 110
Polypus of the uterus, 89
Pyer's patches, tubercular ulcers of, 106
Pyo-pneumothorax dependent on phthisis, 204
Pyo-salpinx, 143, 150
    tumor formed by, 79

RENAL calculus, 119
Rupture of an artery and submucosa in the stomach, 176
    of the heart, 195

SALPINGITIS, syphilitic, 101
Sarcoma, angio-, of the lung, 194
        mesenteric glands in a dog, 202
    of the mesenteric glands in a dog, 80
Sciatic nerve, neuroma of, after amputation of the thigh, 175
Senile gangrene of the ankle, 173
Septic endocarditis, 190
Septicæmia of the new-born, 181
Skull, fracture of the base of, 77
Spinal cord, hemorrhage into the substance of, 178
    secondary degeneration of, 108
Spleen, calcareous concretions of, 101
    enlarged, 123
Splenitis, chronic, 179
        indurative, 181
Staining fluid, new, 107
Stomach, acute atrophy of, 137
    carcinoma of, 157
    contraction of, 80
    myoma of, in a dog, 80
    rupture of an artery and submucosa of, 176
Strangulated bubonocele, 171
Stricture of the œsophagus, 80, 99
    of the urethra, 77
Syphilitic salpingitis, 101

THROMBUS, globular, of Laennec, 136
Tongue, cleft of, 199
Transfusion of a saline fluid, 91
Tricuspid valves, insufficiency of, 91
Tubercle developing in a bronchial gland, 121
Tubercular meningitis, 170
    nodule in flocculus of the cerebellum, 106
    peritonitis, 108
Tuberculosis of the bile-ducts with dilatation, 125
    of the brain, 146
    general, 105, 125
    of the mesenteric glands, 106
    of the pharynx, 88
    primary, of the kidney, 190

UMBILICAL hernia, congenital, 154
Ureters, dilated, 87
        in a fœtus, 87
    multiple cysts of, 87
Urethra, stricture of, 77
Uterus, carcinoma of, 88, 90, 147, 116
    fibroma of, 104, 183
        in a dog, 80
    fungosities of, 168
    pendulous myoma of, 161
    sloughing fibroma of, 88
    polypus of, 89

VALVES of the heart, insufficiency of, 91
Verrucous endocarditis, 202
Vertebræ, cervical, fracture of, 178
Vertebral caries, 83

WOUND, gunshot, of the brain, 91
        of the liver, 115
    stab, of the lung, 132

SERIAL

RB
1
N32
1887

New York Pathological Society
Proceedings

GERSTS